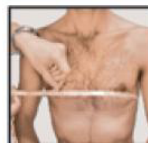




Handbook on Clinical Approach to **Respiratory Medicine**



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Handbook on Clinical Approach to Respiratory Medicine

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PREFACE

Handbook on Clinical Approach to Respiratory Medicine is published with the intention of reaching out to the undergraduates, interns and postgraduates to understand the importance of proper case taking. Medicine is all about knowledge and observation. The essential element is the decisional process by probing history, recognition of signs and clinical reasoning. Hands, ears and the brain play a major role in diagnosing in spite of the laboratory tools available. As scientists, we are on a mission to alleviate the patients' sufferings and have to go deeper into every aspect of the patient's disease process.

The art of history taking and proper examination of the patient has become almost extinct. This book confines basically to case taking in respiratory medicine and will give the reader a deeper insight into history taking and examination of the patient. This methodology will help the reader in examining the other systems like CVS, CNS, etc. Without a correct diagnosis, all our efforts will be futile to treat the patient.

K Surendra Menon

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INTRODUCTION

The content of the book emphasizes on the correct way of history taking, examination of the patient, relevant investigations and arriving at a final diagnosis.

History taking is the time when the doctor and the patient get to know each other and the patient's fears and concerns can be understood. The skills in taking history develop with experience; so, students are encouraged to take history independently. Difficult diagnostic problems are more often solved by carefully taken history than by a battery of laboratory tests. At the end of history taking, the examiner may be able to arrive at a few differential diagnoses before even touching the patient. Our studies have shown that the correct history taking will make us zero in, onto a possible diagnosis in nearly 80% of the cases.

From examination point of view, long case includes history taking and examination of the patient. All systems are required to be examined. So, a quick history taking without missing any relevant points is essential and the 'last minute panic' can be avoided. It is advisable to finish the history taking in 10–12 minutes. But remember this pattern has to be followed after graduation also to become a successful clinician.

Symptoms narrated by the patient should be carefully listened to and as a 'medical detective' ask pertinent questions as some patients may go off the track. Patient's complaints should be documented and analyzed. Like a perfect diplomat, we should show tactfulness and patience when the subject mentions the complaint.

Anatomical Principles

IMPORTANT ANATOMICAL LANDMARKS

One should have a clear understanding of anatomy of the respiratory system to perform a proper physical examination. Some of the important anatomical landmarks are outlined below.

The upper respiratory tract starts from the mouth or nose and includes all the structures in the mouth, nose and sinus up to the larynx (the details of which will be discussed later). Lower respiratory tract, starts from the lower border of cricoid cartilage which includes the trachea, airways and lung parenchyma containing alveoli.

Trachea

1. Starts from cricoid cartilage (lower border of larynx at the level of 6th cervical vertebra posteriorly) to sternal angle anteriorly (angle of Louis) and T5 spinous process posteriorly, where it divides into left and right main stem bronchi. The inner diameter is 25 mm and the length is about 10–16 cm.
2. Trachea is generally in the midline but slight deviation to right may occur in normal individuals (by right aortic arch

and weight of right lung). The weight of right adult lung ranges from 375 to 550 g and the left lung 325 to 450 g.

3. Trachea has intra- and extrathoracic components. This has important bearing in the understanding of physiology of variable obstruction.

The spine of 7th cervical vertebra is an important landmark because of its prominence and thoracic spines can be counted below it.

Angle of Louis

- Angle of Louis is the angle between the body of sternum and manubrium. Many important landmarks occur at this level:
 - 2nd rib articulates with manubrium sterni at this site. The ribs are counted anteriorly starting from this point
 - Carina of trachea is at this level and it branches into right and left bronchi
 - Mediastinum is divided into superior and inferior at this level.

With the help of surface anatomy, the thorax is arbitrarily divided into various spaces and lines. Description of abnormal signs in relation to the ribs, intercostal spaces (areas) and lines help to localize the lesion anatomically (upper lobe, lower lobe, middle lobe, etc.).

Ribs

Anteriorly ribs are counted down starting from 2nd rib. There are 12 ribs and 11 interspaces in each hemithorax. You can also count up from 12th rib. Inferior angle of scapula overlies 7th thoracic rib posteriorly.

Spaces/Areas (Chest Topography)

Anteriorly the spaces are supraclavicular, infraclavicular and mammary and Traube's space (Fig. 1.1).

Supraclavicular: Space above clavicle.

Infraclavicular: Space below clavicle up to 2nd intercostal space.

Mammary: Space below infraclavicular area, i.e. 2nd to 6th intercostal space.

Traube's (semilunar) space: It is a crescent-shaped space, encompassed by the lower border of the left lung, the anterior border of the spleen, the left costal margin and the inferior margin of the left lobe of the liver. Thus, its surface markings are respectively the left 6th rib superiorly, the left anterior axillary line laterally and the left costal margin inferiorly. In

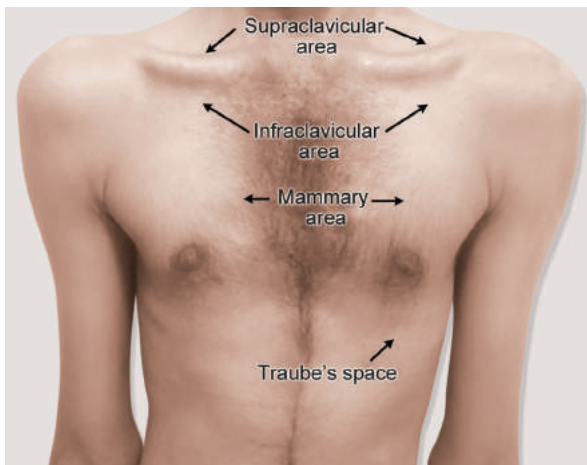


Fig. 1.1: Chest topography—spaces/area

other words left 6th rib in the midclavicular line to 8th costal cartilage in the parasternal line, then along the left costal margin to the 11th rib in the midaxillary line and then the 9th rib in midaxillary line.

Laterally, the spaces are axillary and infra-axillary spaces bound in between by two axillary folds. Anterior axillary fold is formed by pectoralis major muscle and posterior axillary fold by latissimus dorsi and teres major muscles.

- *Axillary area*: Space up to 5th intercostal space in mid-axillary line (on the right where the horizontal fissure meets the oblique fissure)
- *Infra-axillary area*: Below the 5th intercostal space to 7th intercostal space.

Posteriorly, the spaces are suprascapular, interscapular and infrascapular spaces (Fig. 1.2).

- *Suprascapular area*: From the apex to the spine of scapula

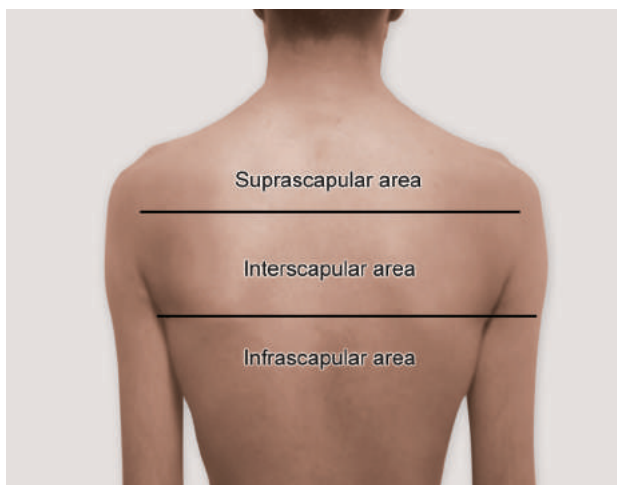


Fig. 1.2: Posterior spaces

- Interscapular area: From the spine of scapula to the angle of scapula
- Infrascapular area: From the angle of scapula to the 11th rib.

Lines

Following are the imaginary vertical lines in the chest: Midsternal, parasternal, midclavicular, anterior axillary, midaxillary, posterior axillary, infrascapular and vertebral lines (Figs 1.3 to 1.5).

Midsternal line: A vertical line down the middle of sternum.

Parasternal line: A vertical line along lateral edges of sternum.

Midclavicular line: A vertical line from midpoint of clavicle.

Anterior axillary line: A vertical line along anterior axillary fold.

Midaxillary line: A vertical line at midpoint between anterior and posterior axillary line. There is less muscle attachment

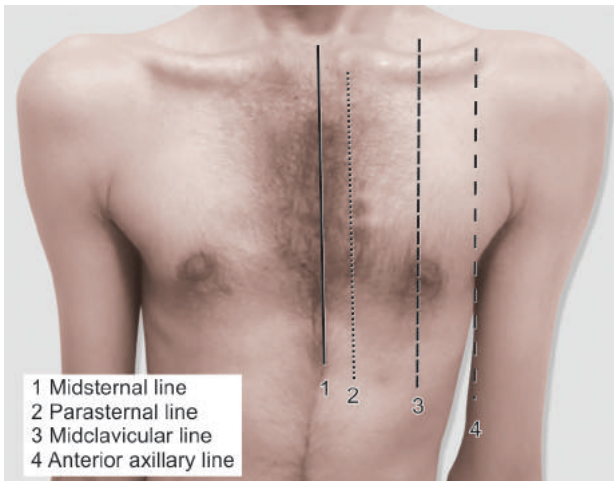


Fig. 1.3: Chest topography—important lines on anterior chest wall

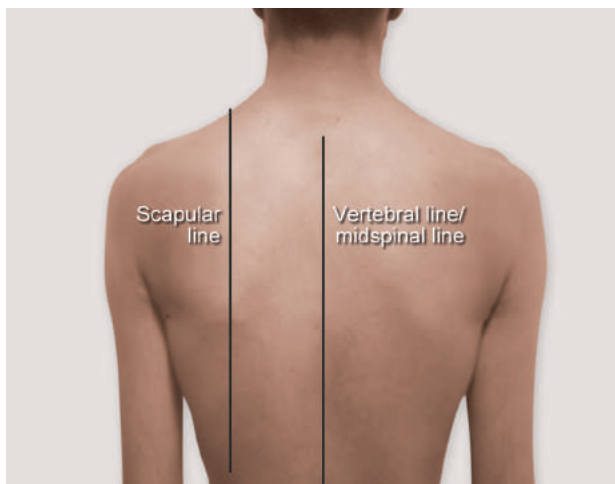


Fig. 1.4: Important lines on posterior chest

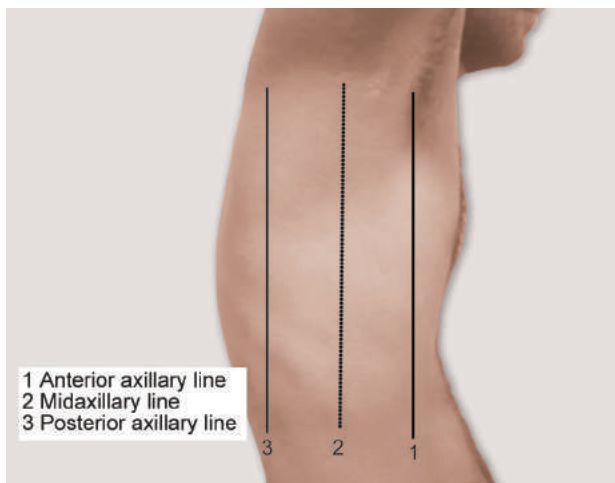


Fig. 1.5: Important lines on lateral chest wall

here, so it is the ideal place for putting intercostals drain and for thoracoscopy incision. This is also called '*Safe triangle*' because there is less risk of injuring blood vessels like internal mammary artery, and damage to muscle and breast tissue.

Safe triangle: It is formed anteriorly by the lateral border of pectoralis major muscle, laterally by the lateral border of latissimus dorsi muscle, inferiorly by the line of 5th intercostal space and superiorly by the axilla.

Posterior axillary line: Along posterior axillary fold.

Scapular line: Starting from the suprascapular area passing down the inferior angle of scapula.

Vertebral line: Over spinous processes in the midline.

Surface Anatomy of Lungs

Right Lung

With a marking pen, start 3 cm above clavicle in midclavicular line, come down along right parasternal line, to join 6th rib in midclavicular line, to 8th rib in midaxillary line and to 10th rib posteriorly. Posterior marking of lung is from the apex to 10th thoracic vertebra posteriorly.

Left Lung

Start 3 cm above clavicle in midclavicular line and draw a line going downwards along the parasternal margin up to 4th costal cartilage. Between 4th and 6th costal cartilage deviate to left by 4 cm. The lower levels of lung—6th rib in the midclavicular line, 8th rib in the midaxillary line and 10th rib in the scapular line.

Kronig's Isthmus

An area about 5–7 cm in the supraclavicular region. It is bound medially by scalenus muscle, laterally by acromion process

of scapula, anteriorly by clavicle and posteriorly by trapezius muscle.

Clinical Significance

Normal percussion over Kronig's isthmus is resonant. The percussion note becomes hyperresonant in emphysema and impaired in early tuberculosis and apical lung tumors.

Surface Anatomy of Lobes (Figs 1.6 to 1.8)

Oblique fissure extends on the left from the tip of spinous process of the T3 vertebra extends down to the level of sixth costochondral junction anteriorly. In taking this route, the approximate path of the 6th rib is followed. The posterior origin of the fissure on the right is slightly inferior—the inferior margin of the T4 vertebra. If the subject keeps his hands on the head, the line drawn from the spinous process of the above vertebrae

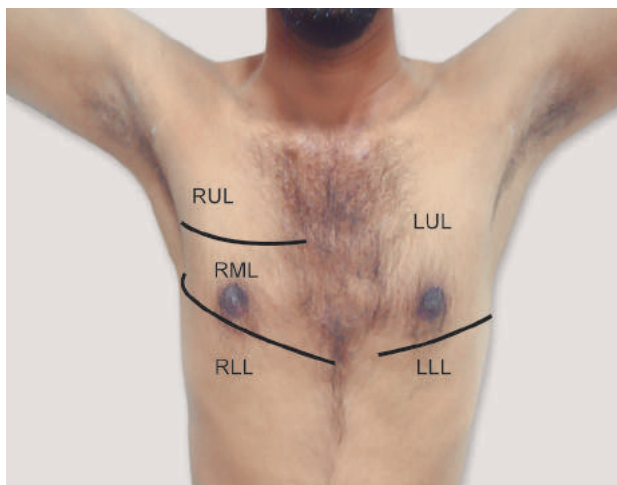


Fig. 1.6: Anterior surface markings of the lung lobes

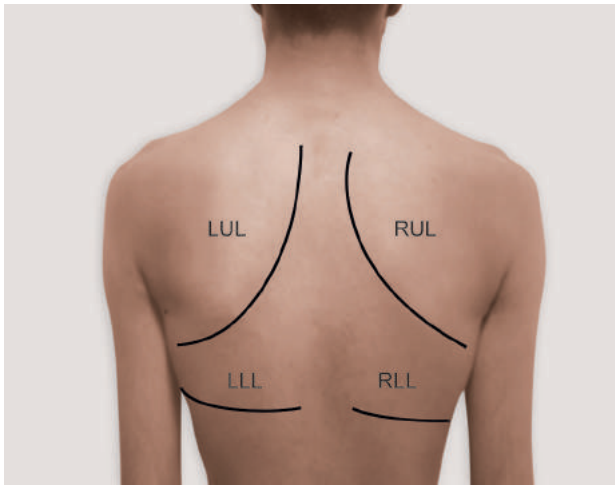
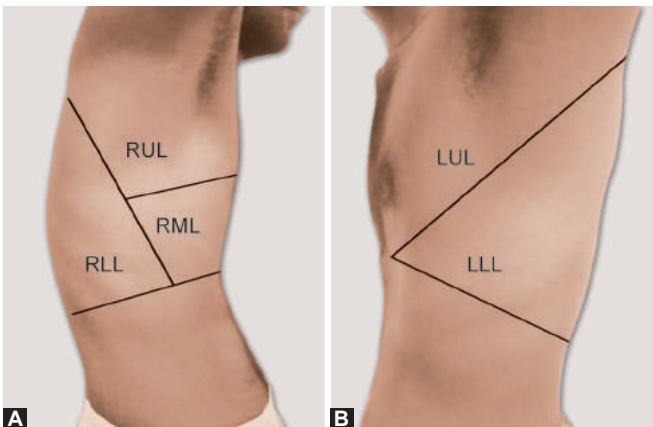


Fig. 1.7: Posterior surface markings of the lung lobes



Figs 1.8 A and B: Lateral surface markings of the lung lobes
(A) Right lung; (B) Left lung

T4 on the right and T3 on the left along the lower border of the scapula to the level of 6th costochondral junction.

Transverse fissure start from the level of the right 4th costal cartilage horizontally to a junction with the oblique fissure at approximately the midaxillary line in the 5th intercostal space.

Remember there are three lobes on the right side and two lobes on the left side normally.

Once the fissures are drawn, one can easily recognize the surface anatomy of lobes of lungs. One can then appreciate the importance of examining the patient in all anatomical areas mentioned above to identify the lobes. Most of the lower lobe is in back, the upper lobe is in front and the middle lobe is in front and in the axilla all the three (right) lobes are present.

Bronchopulmonary Segments

It is a wedge of lung tissue supplied by a single bronchus and corresponding pulmonary artery and vein. There are ten segments in right lung and eight segments in left lung (Table 1.1).

Table 1.1: Bronchopulmonary segments

<i>Right lung</i>	<i>Left lung</i>
Right upper lobe bronchus	Left upper lobe bronchus
Apical segment (1)	Apico-posterior segment (1 and 2)
Posterior segment (2)	Anterior segment (3)
Anterior segment (3)	
Right middle lobe bronchus	Lingula
Lateral segment (4)	Superior segment (4)
Medial segment (5)	Inferior segment (5)

Contd...

Contd...

Right lung	Left lung
Right middle lobe bronchus	Lingula
Apical (superior) segment (6)	Apical (6)
Medial basal segment (7)	
Anterior basal segment (8)	Anterior basal segment (8)
Lateral basal segment (9)	Lateral basal (9)
Posterior basal segment (10)	Posterior basal (10)

No medial basal segment in left lung

Muscles of Respiration

There are two types of muscles of respiration—principal and accessory muscles.

Principal muscles are used during normal inspiration while accessory muscles are used during forced breathing (heavy exercise and exacerbations of obstructive airway diseases).

Principal muscles include external intercostals, intrachondral part of internal intercostals and diaphragm.

The width of the thoracic cavity (lateral and anteroposterior diameter) is increased by external and intrachondral part of internal intercostals which elevate the ribs. The lateral dimensions of the thorax are increased by the bucket handle movement of the ribs. Diaphragm contracts to increase the vertical dimensions of thoracic cavity and also aids in elevation of lower ribs and abdominal contents are pushed downwards.

Accessory muscles of inspiration are sternocleidomastoid which elevates the sternum and scalene muscles (anterior, middle and posterior) elevate the first two ribs.

Remember to look for alae nasi when the patient is in respiratory distress that means a fall in FEV_1 by 30%.

Muscles of expiration: It is passive as no muscle involvement in normal breathing. The process is simply done by elastic recoil of the lungs and the rib cage.

But during active breathing (exercise), the intrachondral part of the internal intercostal muscles assist active expiration by pulling the ribs downwards and inwards. They also prevent bulging of intercostal spaces during straining like vigorous coughing and vomiting. The abdominal muscles when contract (rectus abdominis, external oblique, internal oblique and transversus abdominis), intra-abdominal pressure increases and push the diaphragm upwards and force the air out of the lungs.

Diaphragm (Fig. 1.9)

The diaphragm is a dome-shaped structure containing muscle and fibrous tissue that separates the thoracic cavity from the abdomen. The dome curves upwards. The superior surface of the dome forms the floor of the thoracic cavity and the inferior surface the roof of the abdominal cavity.

As a dome, the diaphragm has peripheral attachments to structures that make up the abdominal and chest walls. The muscle fibers from these attachments converge in a central

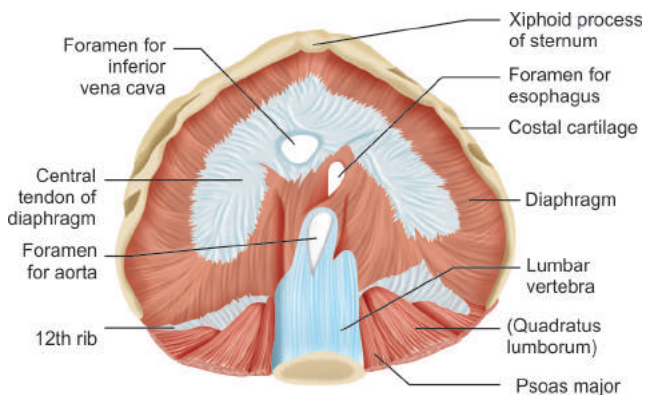


Fig. 1.9: Diaphragm

tendon, which forms the crest of the dome. Its peripheral part consists of muscular fibers that take origin from the circumference of the inferior thoracic aperture and converge to be inserted into a central tendon.

The muscle fibers of the diaphragm emerge from many surrounding structures. Anteriorly, fibers emerge from behind the xiphoid process and the cartilages of the floating ribs, (ribs 7–12). Laterally, fibers emerge from the sides of the ribs themselves, including the two false ribs 11 and 12. Posteriorly, fibers emerge from the abdominal wall and lumbar vertebrae.

There are two lumbocostal arches, medially and laterally.

Crura and central tendon: The left and right crura are tendinous in structure, and blend with the anterior longitudinal ligament of the vertebral column.

The central tendon of the diaphragm is a thin but strong aponeurosis situated near the center of the vault formed by the muscle, but somewhat closer to the front than to the back of the thorax, so that the posterior muscular fibers are the longer. Once the diaphragm has been outlined, you can appreciate that the pleural gutter is deep posteriorly. Fluid thus tends to accumulate posteriorly.

Mediastinum (Fig. 1.10)

Mediastinum is the space between the lungs from inlet to outlet of thorax. Superiorly, by suprasternal notch. Anteriorly, it is in between parasternal lines. Posteriorly, by vertebral line. Inferiorly, it extends to xiphisternum. Mediastinum is narrow posteriorly and widens anteriorly. Since the inlet of thorax is slanted, only posterior mediastinum extends to neck.

Sternal angle separates superior from inferior mediastinum. The inferior mediastinum is divided into anterior, middle and

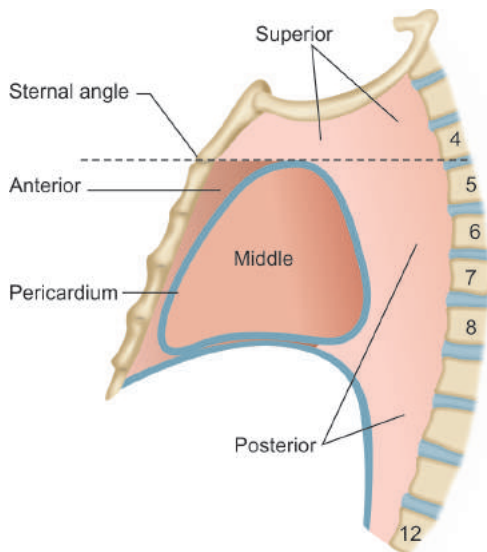


Fig. 1.10: Mediastinum

posterior compartments. The space in front of the heart is anterior mediastinum and behind is posterior mediastinum. Heart itself defines the middle mediastinum. The posterior mediastinum is divided into two, i.e. paravertebral and prevertebral space. Superior mediastinum extends into the neck and is called cervico-mediastinal space.

It is important to know the structures in each compartment. It is important to differentiate the masses in the mediastinum in relation to the structures there.

Pleura and Pleural Sacs

Parietal and visceral pleura cover the surface of the lung. Parietal pleura that lines inner surface of rib cage and outer

portion of each hemi diaphragm is supplied by intercostal nerves and this is one reason for localized chest pain.

The pleura in the central region of each hemidiaphragm is innervated by fibers that travel with the phrenic nerve (C3-C4). So, in infection involving this area, the pain extends to ipsilateral neck or shoulder even to the abdomen presenting as acute abdomen. Parietal pleura has innervations and is pain sensitive but not the visceral pleura.

The pleural sac extends to 8th, 10th and 12th rib in the midclavicular line, the midaxillary line and scapular line respectively at the lower level. There is a difference in the pleural reflection on the left side because the lung and the pleura from midsternal line lies at a higher level, i.e. at the lower border of the 4th costal cartilage. The superficial cardiac dullness is obtained in this area because the heart is uncovered.

Hilum

Hilum lies opposite to the spines of 4th, 5th and 6th thoracic vertebrae. Left hilum is at a higher level.

Costal Angle

Costal angle (subcostal angle) is formed by the 10th rib with costal cartilage on both sides and xiphisternum in the middle. The normal angle is not more than 90° but more acute in males. Both the sides are symmetrical. Volume changes in each hemithorax will alter this relationship. Hyperinflated lungs will increase the costal angle. In alar chest anterior-posterior distance is reduced. Diaphragmatic paralysis also alters the symmetry of costal angle.

Spinous Process

The most prominent spinous process is of 7th cervical vertebra. You can count down the thoracic vertebra and the ribs using this landmark.

Symptomatology

CARDINAL RULE TO HISTORY TAKING

The cardinal rule while presenting the history is “To avoid medical terms (one has to use the patients’ words).”

The diseases of respiratory system may affect airways, lung, parenchyma, pleura and the mediastinum.

Final diagnosis in respiratory medicine always has three parts (systemic disease to be excluded) that are as follows:

1. Anatomical: For example, right upper lobe, left lower lobe, pleura, etc.
2. Pathological: For example, fibrosis, bronchiectasis, cavity, effusion, pneumothorax, consolidation, etc.
3. Etiological: For example, tuberculosis, bacterial or viral pneumonias, etc.

When diagnosis is made, it should have the above three parts and if the patient is having any associated systemic illness like hypertension, diabetes mellitus it has to be mentioned to make it a complete final diagnosis.

For example: Right upper lobe bronchiectasis, post-tubercular with diabetes mellitus.

Common Symptoms

Common Respiratory Symptoms

- Cough—dry or wet (Expectoration)
- Hemoptysis
- Breathlessness (Dyspnea)
- Chest pain
- Hoarseness of voice
- Wheezing
- Stridor
- Sleep disturbances
- Dysphagia
- Sneezing, rhinitis
- Headache
- Allergy to certain food, flowers, dust, etc.

Constitutional Symptoms

- Fever
- Loss of weight
- Loss of appetite
- When the patient walks in, observe the patient (breathlessness, gait, facial expression, etc.). Ask the patient to be seated or make him lie down if he is very sick.

Before the patient gives his complaints, note the following:

Proper History Taking

<i>Name</i>	:
<i>Age</i>	: Older age group: Chronic bronchitis, lung cancer, pulmonary tuberculosis.
<i>Younger age group</i>	: Pulmonary tuberculosis, asthma
<i>Sex</i>	: COPD, carcinoma bronchus more in males.

	Eventration of diaphragm, Idiopathic Pulmonary Arterial Hypertension (IPAH), Lymphangioleiomyomatosis (LAM) more in females.
<i>Occupation</i>	: Disease like silicosis, asbestosis, byssinosis, hypersensitivity pneumonitis.
<i>Race</i>	: Asians are more prone to tuberculosis.
<i>Recent history of travel</i>	: Diseases in endemic areas (parasitic infections).
<i>Residential address</i>	: Near a factory, slum residents (environmental factors).

How to Proceed Further

Presenting complaints (chief complaints)—note down the complaints in chronological order.

History of presenting illness—details (elaboration) of the above given symptoms in patient's words.

- Past history
- Family history
- Personal history
- Treatment history
- Menstrual history in females.

SYMPTOMS OF RESPIRATORY DISEASES

Common Respiratory Symptoms

Cough

Cough is the most common respiratory symptom. It is one of the most important defensive functions that protects the lower respiratory tract by preventing foreign material entering from

outside and preventing stagnation of the secretions within the tract (pooling of secretions can lead to secondary infections and worsening of existing condition like bronchiectasis). Coughing is a response to any irritating stimuli from the level of pharynx down. By coughing, the irritants can be forcefully expelled. Coughing is a sudden expiration with closure of vocal cords. Cough reflex occurs as a result of the stimulation of the airway receptors.

Various types of cough have been described. (Common is dry or wet cough)

- a. Hacking dry cough—pharyngitis, smoker's cough.
- b. Barking cough—croup, hysteria.
- c. Brassy or metallic cough—mediastinal tumors, aortic aneurysm.
- d. Paroxysmal cough.
- e. Bovine cough—affliction of vocal cord.

Any cough lasting more than 2 weeks should be investigated. Worsening of cough in chronic bronchitis may be due to underlying lung malignancy. Sometimes, a bout of cough may produce sudden and transient loss of consciousness (cough syncope). Common causes and characteristics of cough are discussed in Table 2.1.

Table 2.1: Common causes and characteristics of cough

Common causes	Nature/character
1. Postnasal drip, usually irritate pharynx (now called as upper airway cough syndrome, UACS)	Usually persistent
2. Laryngitis, whooping cough, croup, tumor	Harsh, barking, painful, persistent associated with laryngeal tumor
3. Tracheitis	Painful
4. Bronchitis (acute and chronic)	Dry or productive, scanty or more in the morning

Contd...

Contd...

Common causes	Nature/character
5. Asthma	Dry or productive, more at night or after exposure to cold air, allergens
6. Pulmonary tuberculosis	Associated with fever, loss of weight
7. Carcinoma bronchus	Persistent, often with hemoptysis
8. Bronchiectasis	Purulent, large quantity, occasional hemoptysis, postural dependence, foul smell
9. Lung abscess	Same as bronchiectasis
10. Pneumonia	Dry initially, later productive
11. Pulmonary edema	Often at night; may be productive with pink frothy sputum.
12. Interstitial lung disease	Dry irritating and distressing
13. Drug-induced (ACE inhibitor/ β blocker)	Dry
14. GERD	Dry

Cough can be dry or productive (with sputum production).

Dry Cough

No sputum production and the cough is irritating to the patient. Some of the conditions are mentioned above. Exposure to dust, postnasal drip, chronic pharyngitis, chronic laryngitis, foreign body, inhalation of irritant gases, smokers' cough, chronic sinusitis, foreign body or wax in auditory meatus. Pressure on the airways as in aortic aneurysm, dilated left atrium, enlargement of mediastinal lymph nodes, retrosternal thyroid and drug-induced cough may be the other causes. In hysterical cough, there is no sputum production and the patient's attention is diverted (like asking rapid fire questions), the cough ceases and patient will be able to speak normally.

Cough with Expectoration

- 1. Quantity:** Normally 10–20 mL of mucous is produced daily as it is a protective mechanism. This will help in

ciliary movement which will help in clearing the airways of unwanted elements. Excess production will stimulate the respiratory mucous membrane and it will be expectorated out. It is better to encourage the patient to use the sputum cup so that the patient will not spit in the open. Usually, the quantity is assessed in cup measurement (standard cup is approximately 200 mL). While taking ward rounds or during case taking it is worthwhile to have a look at the sputum cup (opened!) which will yield a lot of information.

Large quantities are seen in lung abscess, bronchiectasis, etc. Three layered sputum can be demonstrated in the above conditions. The patient is asked to expectorate out the sputum in a glass jar and after 24 hour we may be able to appreciate three layers of sputum—the top most layer will be mucoid, middle layer mucopurulent and bottom layer purulent. In olden days, importance was given to this observation, now replaced by X-rays and CT scans. In bronchoalveolar cell carcinoma, the quantity may be 2–3 liters per 24 hours called bronchorrhea (how distressing!). In early stages of pulmonary tuberculosis, pneumonia, acute and chronic bronchitis, the sputum may be scanty.

2. Color and appearance:

<i>Layman's term</i>	<i>Medical Jargon</i>
White	Mucoid
Yellowish white	Mucopurulent indicates infection. Antibiotics may be given
Yellowish	Purulent. Definite infection. Antibiotics should be given

Infections can precipitate exacerbation of asthma, cor pulmonale, worsening of chronic bronchitis, bronchiectasis, etc. Here antibiotics are definitely indicated without any delay.

Green color: It commonly indicates *Pseudomonas* infections. Sometimes normal individuals expectorate greenish sputum in the morning. It is due to the breakdown of leucocytes in the night and subsequent release of verdo-peroxidase enzyme which gives the greenish color to the sputum. Unlike *Pseudomonas* infection, no treatment is required.

Rusty color: In pneumonia, especially streptococcal pneumonia, during the stage of red hepatization, sputum has rusty color because of the destruction of the RBCs.

Anchovy sauce: Hepatopulmonary amoebiasis

Red currant jelly: *Klebsiella pneumonia*

Pink and frothy: Pulmonary edema

Black or gray: Smokers, industrial and atmospheric pollution, coal miners' disease (pneumoconiosis), bacterial or fungal infections (nocardiosis, aspergillosis)

Hemoptysis: Will be discussed later.

3. Consistency:

- Thick and difficult to expectorate (chronic bronchitis)
- Thin and watery (asthma).

4. Effect on changing position (Postural variation):

Patients with suppurative lung diseases like bronchiectasis, lung abscess, etc. will have increased cough and sputum production with change in postures. So they tend to adopt a position least distressing to them. For example, a patient with left-sided lesion prefers to lie on the same side and if he is asked to turn to right side he will start coughing and bring out sputum. Because of the gravity, the secretions come to the major airways where the cough receptors are situated and patient coughs out the sputum thus preventing pooling of secretions and secondary infection. We would like the patient to lie on the opposite side of the lesion (here right side) to clear away the secretions and postural

drainage with the help of a physiotherapist, so as to prevent secondary infection.

5. Associated chest pain while coughing. (Discussed later).

6. Foul smelling sputum: It is due to anaerobic organisms (*Peptostreptococcus*, *Fusobacterium*, *Bacteroides*) which produce short chain fatty acids—butyrate or butyric acid. Seasonal and diurnal variation should be inquired.

Hemoptysis

Coughing out blood. It may be a large quantity (aspergilloma), bronchiectasis or streaking of sputum (carcinoma bronchus initial stage).

To remember—the bleeding may not be necessarily from the lower respiratory tract. It may be from nose (epistaxis), gums (gingivitis) leading to irritation of larynx or pharynx as postnasal drip or UACS.

Causes:

Respiratory

- Pulmonary tuberculosis
- Carcinoma bronchus
- Bronchiectasis—especially bronchiectasis sicca (dry bronchiectasis) where the patient will not have recurrent respiratory infections, but occasional hemoptysis. The lesion in the upper lobes have no pooling of secretions because of gravity
- Aspergilloma
- Foreign body
- Lung abscess
- Benign lung tumors
- Trauma
- Pulmonary infarction
- Catamenial hemoptysis (occurs during menstruation).

Cardiovascular

- Mitral stenosis
- Pulmonary edema (pink frothy sputum).

Others

- Blood dyscrasias.

Hemoptysis—quantification

- Massive hemoptysis—600 mL or more in 24 hours as one episode or at different bouts or continuous hemoptysis of more than 100 mL/hour for 3 hours or any amount which causes hemodynamic instability
- Moderate hemoptysis—50–200 mL in 24 hours
- Minimal hemoptysis—streaking of sputum with blood (carcinoma bronchus is an example) or 50 mL or less in 24 hours.

Most important cause of death in hemoptysis is due to aspiration and asphyxia rather than exsanguination. The difference between hemoptysis and hematemesis are discussed below in Table 2.2.

Table 2.2: Differences between hemoptysis and hematemesis

<i>Hemoptysis</i>	<i>Hematemesis</i>
Coughing out blood	Vomiting blood
Bright red	Coffee-colored or dark brown
Frothy	Mixed with food particles
Small quantity (not essential)	Large quantity
Past history of respiratory disorder	Past history of gastrointestinal disorder
Malena absent	Malena present
pH alkaline	pH acidic
Bronchoscopy	Upper GI endoscopy

This differentiation is not the rule of thumb. Overlap can occur but still it is a useful guide. A detailed history taking is useful in distinguishing the two. Hemoptysis should be taken seriously even if it is minimal and investigated.

Spurious hemoptysis

From upper respiratory tract (epistaxis, gingivitis, etc.) producing postnasal drip (UACS).

Pseudohemoptysis

Due to pigment prodigiosin produced by gram negative organism—*Serratia marcescens*.

Endemic hemoptysis

Produced by *Paragonimus westermani* (Lung fluke).

Breathlessness or Dyspnea

When presenting the history, breathlessness only can be used as it is the layman's term (not dyspnea). Inquire about any relation to seasonal or diurnal variation.

Dyspnea is the unpleasant awareness of one's own breathing.

Various grading systems are available—MMRC (commonly used), New York Heart Association (NYHA). For simplicity and understanding the following classification is useful.

Grading of dyspnea

Grade I: On unaccustomed exertion. For example, everyday a normal person climbs two flights of stairs but if asked to climb four flights he will become breathless. This is an *unaccustomed* exertion.

Grade II: On accustomed exertion. For example, the same person after a few years may not be able to climb two flights at a stretch and he may have to rest in between.

Grade III: Minimal exertion like bathing, walking a few steps, buttoning the shirt, etc. may produce breathlessness.

Grade IV: Dyspnea at rest.

Clinically useful classification

- 1. Dyspnea of sudden onset**—pneumothorax, asthma, pulmonary embolism, pulmonary edema, foreign body in major airways, inhalation of noxious gases.
- 2. Subacute dyspnea**—progressing over weeks or months. Congestive cardiac failure, anemia, obesity, pleural effusion, ascites, pregnancy, interstitial lung disease.
- 3. Chronic dyspnea (progressing over months or years)**—chronic bronchitis, emphysema, pneumoconiosis, pulmonary fibrosis (common in India due to pulmonary tuberculosis).
- 4. Dyspnea on exertion**—anemia, pregnancy, pulmonary thromboembolism, mitral stenosis, chronic bronchitis, asthma, pulmonary fibrosis, kyphoscoliosis, obesity.
- 5. Dyspnea at rest**
 - Acute mechanical or infective condition
 - Pneumothorax, pleural effusion, pulmonary infarction, pneumonia
 - Paroxysmal dyspnea
 - Pulmonary edema, asthma
 - Psychogenic
 - Hyperventilatory syndrome
 - Metabolic
 - Acidosis of uremia or diabetes (Kussmaul breathing)
 - Cheyne-Stokes breathing (hyperpnea and apnea) (as in cerebrovascular accidents)
 - Biot's breathing
 - Ondine's curse.
- 6. Orthopnea**—the patient is more comfortable sitting up and may become breathless on lying flat. Usually occurs in cardiac failure due to increase in left atrial pressure.
- 7. Paroxysmal nocturnal dyspnea**—as the term indicates breathlessness occurs at night (early morning), pulmonary edema (previously known as cardiac asthma) and asthma.

- 8. Platypnea**—shortness of breath in upright position—arteriovenous malformation in lung as occurs in chronic liver disease (hepatopulmonary syndrome) or hereditary condition—atrial myxoma.
- 9. Orthodeoxia**—oxygen desaturation in upright position which is more than 3%.
- 10. Trepopnea**—breathlessness while lying on the side—cardiac causes, unilateral massive pleural effusion.

Chest Pain

Pain in the anterior part of the chest is an important symptom in respiratory and cardiac disorders and to be taken seriously.

When the patient complains of chest pain, a detailed history taking is important. It may be either due to conditions like *cellulitis, mastitis, premenstrual pain, myalgia, herpes, intercostal neuralgia, rib fractures, costochondritis* (Tietze's syndrome) or due to serious causes like *angina, myocardial infarction, pleuritic pain, gastroesophageal reflux disease, rib erosions, mediastinitis*, etc.

As far as this book is concerned, emphasis is given to chest pain secondary to pulmonary disorders. For example, pleurisy, pneumothorax, acute pulmonary embolism, pneumonia, massive collapse of the lung, lung abscess, bronchiectasis, carcinoma bronchus, pneumomediastinum, mediastinal growth, etc.

Pleuritic pain is usually localized (not diffuse), sharp and stabbing, dull ache or felt as a 'catch', aggravated by coughing, sneezing, laughing, crying and deep breathing usually felt at the end of inspiration caused by inflamed pleura. The innervation of pleura is discussed earlier.

Patients with pleuritic pain often prefer lying on the side of pain or support the affected area with the hand (auto-splinting).

Recurrent pleuritic pain can occur in bronchiectasis during infection.

Hoarseness of Voice

Patient notices change in voice. Common causes are pharyngitis, laryngitis, tuberculous laryngitis, carcinoma larynx, recurrent laryngeal nerve palsy, and inhaled corticosteroids producing myopathy of adductor muscles of the vocal cord, Ortner's syndrome.

Wheezing

Some patients may complain of abnormal or whistling noises produced in the chest while breathing as in the case of asthmatics or patients with chronic bronchitis, vocal cord dysfunction.

Stridor

This is a serious condition which results from partial obstruction, narrowing of larynx, trachea or major bronchi usually inspiratory and may be audible without stethoscope.

Sleep Disturbances

Inquire about the patient's sleep pattern, snoring history and history of apneas—usually elicited from the bed partner.

Other Symptoms

- **Dysphagia:** Compression of esophagus secondary to enlarged mediastinal node compression due to various pathological conditions (cannot include as a respiratory symptom exclusively)
- History of sneezing and rhinitis (seasonal variation), allergy to food, pollen, etc.

Constitutional Symptoms

Fever

Normal temperature is 36.6–37.2°C when taken orally, rectal temperature is 0.5–0.6°C higher but axillary temperature is 0.5°C less. Usually rise in temperature above 37.2°C in the morning and 37.7°C in the evening is taken as pyrexia. All individuals have a circadian rhythm with evening temperature of 0.5–1°C more than in the morning. A slight rise in temperature during ovulation can occur.

Types of Fever

1. Continuous: Temperature is present throughout the day but never touches the baseline in 24 hours and the fluctuation is not more than 1°C.
2. Remittent: Temperature never touches the baseline in 24 hours and the fluctuation is more than 2°C.
3. Intermittent: Temperature touches the baseline at least once in 24 hours.

Note: Evening rise of temperature described in tuberculosis is because of the increased levels of cytokines (IL-1).

Loss of Appetite

Patient may not be eating well now as used to be earlier and they may have disinclination to take the food.

Loss of Weight

Patient or the relatives have noticed the loss of weight. In India, two most common causes for loss of weight are pulmonary tuberculosis or carcinoma bronchus.

Significant weight loss

(Different authors give different percentage)

- 5% in 30 days

- 7.5% in 60 days
- 10% in 180 days.

HISTORY TAKING

Past History

Previous history of any illness might give us a clue regarding the present disease.

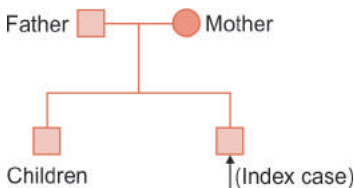
In respiratory medicine: **the must to know** are past history of asthma, allergic disorders, diabetes mellitus, hypertension, pulmonary tuberculosis (contact history), epilepsy (aspiration pneumonia), childhood history of measles, whooping cough.

Family History

History of asthma, allergy, diabetes mellitus, hypertension, epilepsy (all this can be inherited) and history of symptoms suggestive of pulmonary tuberculosis in other family members should be elicited.

Personal History

Sleep, bowel and bladder habits, smoking (smoking index is number of cigarettes smoked per day $\times$ number of years), alcohol use, substance abuse, dietary habits, spouse and children (pedigree chart).



Socioeconomic History

Socioeconomic status can be inquired into and can be made out from the dress, cleanliness and occupation.

Treatment History

This may be elicited with the past history but patients with respiratory disorder volunteer even the specific names of the drugs like an asthmatic may say he is on salbutamol, or aerosol therapy or a person who had tuberculosis may give the name of the drugs he had taken or say while taking the drugs urine was orange-colored (rifampicin). History of surgery, anesthesia (aspiration pneumonia) should be taken. History of treatment for hypertension is important because ACE inhibitors produce dry cough.

Menstrual History

Must in all adult female patients, especially LMP (last menstrual period).

One of the most common causes of sterility in India is genitourinary tract tuberculosis. All types of menstrual irregularities can be encountered in genitourinary tuberculosis.

In endometriosis (ectopic endometrial tissue), the patient may have hemoptysis or pneumothorax during menstruation (rare).

Summarizing the History

After completing history taking, try to summarize the history (as some of the examiners will prefer to have only positive points from the history) to come to a provisional diagnosis.

MAKING PROVISIONAL DIAGNOSIS FROM HISTORY OBTAINED

Once the history taking is systematically done, we may be able to come to a differential diagnosis even before touching the patient (in nearly 80%).

Look at the examples given below.

Case Study 1

A 45-year-old manual laborer with smoking habit, complaints of cough with scanty mucopurulent expectoration of 3 weeks duration with occasional hemoptysis for the last 7 days, streaky in nature. He has occasional fever and loss of weight. He was treated at a local hospital but not relieved. He also has history of breathlessness for the last 5 years for which he takes occasional medication.

There is a past history of pulmonary tuberculosis, but no history of asthma, hypertension, diabetes, epilepsy. There is no family history of any of the above conditions. Personal history of smoking (smoking index of 400), not an alcoholic, normal bowel and bladder habits, consumes mixed diet. He is married and has two children. For the above symptoms, he was taking 5 injections from the local doctor with some syrup recently. He was getting treatment at the local hospital for breathlessness and recurrent respiratory infections for the last 5 years.

Differential Diagnosis from History

- a. Pulmonary tuberculosis.
- b. Carcinoma bronchus.
- c. Chronic obstructive pulmonary disease. Increase in frequency of coughing or hemoptysis as an indicator for malignancy.
- d. Bronchiectasis (not the typical symptoms).

Case Study 2

A 50-year-old male patient, a quarry worker was brought to the casualty with coughing out yellowish white sputum, hemoptysis and fever of 5 days duration. Blood was bright red in color and frothy, about 100 mL in 24 hours. He was apparently alright 5 days back. He had no associated complaints. He gave past history of taking antitubercular treatment 3 years back and had completed the full course. There is no past history of asthma, diabetes, hypertension, epilepsy or aspiration. No family history of asthma, diabetes, hypertension or epilepsy.

Personal history—smoker, alcoholic, bowel and bladder habits normal. Consumes mixed diet.

Treatment history of tuberculosis (intermittent regimen), completed 6 months of treatment 3 years back.

Differential Diagnosis from History

- a. Reactivation pulmonary tuberculosis.
- b. Silicotuberculosis.
- c. Bronchiectasis.
- d. Carcinoma bronchus.
- e. Pneumonia.
- f. Lung abscess.

Case Study 3

A 38-year-old asthmatic lady was admitted with the complaints of left-sided chest pain, cough with yellowish sputum, fever and increasing breathlessness in spite of her regular medications since 10 days. On further questioning, she had also complaints of loss of appetite and loss of weight. Her relatives also noticed change in her voice. She is asthmatic from childhood and on regular treatment with metered dose inhalers. No history

of diabetes, hypertension, epilepsy and no history of taking antitubercular treatment.

Family history of asthma, diabetes but no history of hypertension, epilepsy or pulmonary tuberculosis.

Personal history—normal bowel and bladder habits. Normal appetite with fewer intakes now. Married and has one child. Her spouse and child are healthy according to her.

She is using inhaled corticosteroids and long acting β_2 agonist for the last 2 years. Before that she was on oral bronchodilators. Her menstrual cycle is irregular and with excessive bleeding.

Differential Diagnosis from History

- a. Acute severe asthma secondary to infection.
- b. Pneumothorax (left).
- c. Pulmonary tuberculosis. Long history of asthma, already on inhaled steroids and probably would have taken oral corticosteroids knowingly or unknowingly with probable genital tuberculosis (irregular periods).
- d. Hoarseness of voice may be due to acute or chronic pharyngitis, laryngitis, tubercular laryngitis or she was not instructed to wash the mouth and gargle after using inhaled corticosteroids. This may lead to myopathy of adductor muscles of vocal cord.
- e. Pneumonia.
- f. Pleural effusion (left).
- g. To rule out diabetes mellitus (family history of diabetes and probably secondary to chronic steroid use).

Examination of Respiratory System

- Orientation to time, place and person (this will give us an idea about patient's conscious level)
- Build and nourishment
- **Anthropometry:**
 - Height
 - Weight
 - Upper and lower segment ratio
 - Calculation of body mass index (BMI).

Warm your hands before touching the patient and inform the patient what you are going to do.

VITAL SIGNS

- **Temperature:** Normal 36.6–37.2°C. *Temperature was discussed in detail while dealing with symptomatology*
- **Pulse:** Usually radial pulse is palpated. Normal is taken as 70 and 80 beats per minute while resting. Examiner has to check for— rate, rhythm, volume, character, force, condition of arterial wall, radial-femoral delay, examining peripheral pulse, check for left and right radial pulse to know that they are equal (difference may be there in coarctation of aorta, aortitis, peripheral embolism)

- **Respiratory rate:** Assess the following—rate, rhythm, type and use of accessory muscles. Normal—16 to 20 per minute while resting. The respiration pulse ratio is roughly calculated as 1: 4
Inspiration is an active process whereas expiration is passive. The type of respiration in males is usually abdominothoracic and in females it is thoracoabdominal type and in disease conditions, the type may alter (Ref page 54)
- **Blood pressure:** Normal systolic 120 mm of Hg and diastolic is 80 mm of Hg at rest and variation can occur with age. The readings may vary with the time of the day, anxiety, temperature, etc.
- **Pulse oximetry** (now walk pulse oximeter is available)—to measure SpO_2 .

GENERAL PHYSICAL EXAMINATION

Examine for pallor, icterus, cyanosis, lymphadenopathy, clubbing, pedal edema, JVP, any scars on the visible part of the body (this may give us a clue regarding pleural tap, ICD insertion scars, thoracotomy scars, 'branding scars,' etc.).

Pallor

Pallor is very common and can be appreciated when palpebral conjunctiva is examined. It may be due to blood loss secondary to hook worm infestation, severe hemoptysis, hematemesis and menorrhagia. There will be individual variation, so anemia can only diagnosed after getting the hemoglobin report.

Suffused conjunctiva may be due to secondary polycythemia or superior vena cava obstruction.

Icterus

Bulbar conjunctiva is to be examined for yellowish discoloration—icterus, again the interpretation is subjective. So serum bilirubin report will be confirmatory. The patient has to be examined in day light and not artificial light. Muddy conjunctiva can mislead.

Cyanosis

Bluish discoloration of the skin and the mucous membrane due to an increase in the amount of reduced hemoglobin in the capillary bed in excess of 5 grams/100 mL. It is of three types:

Central Cyanosis

Causes

1. Decreased arterial oxygen saturation
 - a. High altitude.
 - b. Impaired pulmonary function (COPD, asthma, diffuse pulmonary lung disease, pneumonia.
 - c. Anatomic shunts—cyanotic congenital heart disease, pulmonary AV fistula and intrapulmonary shunts.
 - d. Hemoglobin with low affinity for oxygen (Hb Kansas-Hb K).
2. Hemoglobin abnormality
 - a. Methemoglobinemia
 - Hereditary
 - Acquired: Drug-induced—nitrates, sulfonamide.
 - b. Sulfhemoglobinemia.
 - c. Carboxyhemoglobinemia (smokers).

Peripheral Cyanosis

Causes

- a. Reduced cardiac output.
- b. Cold exposure.
- c. Redistribution of blood flow from extremities.
- d. Arterial and venous obstruction.

Differential Cyanosis

Causes

- a. PDA with pulmonary arterial hypertension with right to left shunt—cyanosis is seen in lower limbs.
- b. PDA with pulmonary arterial hypertension with right to left shunt and transposition of great vessels—cyanosis is seen in upper limbs.

Differentiating Features between Central and Peripheral Cyanosis

Central Cyanosis

Whole body may be bluish—look at the nails and under the tongue, this is due to right to left shunt or due to diseases of lung. Extremities are warm and may be associated with clubbing or polycythemia. Oxygen inhalation may show marginal improvement and PaO_2 is usually low.

Peripheral Cyanosis

Cold, clammy extremities, with bluish discoloration of nail bed, nose tip, ear lobe. Occurs due to peripheral stasis. Bluish discoloration disappears on warming. No change with oxygen inhalation and PaO_2 is normal.

Lymphadenopathy

It can be generalized or localized. The sites generally looked for presence of node enlargement are supraclavicular,

scalene, axillary, submental, submaxillary, submandibular, preauricular, posterior auricular and inguinal.

When examining for supraclavicular node, the subject is to be seated upright and examined from behind.

For scalene node, place the index finger between clavicle and sternomastoid muscle and press down towards the 1st rib with the head slightly tilted to same side. The node is usually involved secondary to malignancy.

TB lymphadenopathy: Usually affects cervical lymph node but may affect axillary, inguinal, etc. They may be initially firm and freely mobile and later become soft, immobile and matted together and adherent to skin and adjacent structures; sometimes with sinus formation and cold abscess.

Malignancy of lung: Stony hard; usually supraclavicular and scalene node. If axillary node enlargement, suspect chest wall involvement by malignancy.

Virchow's node: Troisier's sign—usually seen in the left supraclavicular fossa. Occurs in carcinoma of the lungs, stomach, kidney or testis.

AIDS: Persistent generalized lymphadenopathy with symmetrical, mobile and nontender. Nodes usually affected are cervical, supraclavicular, submandibular and axillary nodes.

Lymphomas: Rubbery, nontender lymph nodes in the neck.

Clubbing

Enlargement of the terminal phalanges of the digits due to increased subungual soft tissue.

Many theories have been attributed to clubbing like humoral, neurogenic, hypoxia, decreased ferritin and platelet derived growth factor.

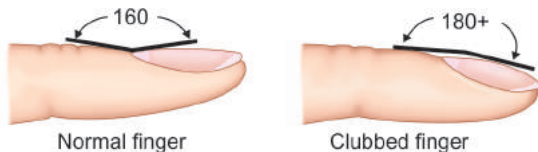


Fig. 3.1: Lovibond's angle

Lovibond's angle: The angle between the nail and nail bed is normally 160° but in clubbing as illustrated in Figure 3.1 will increase.

Grade I: Increased glossiness of skin over the nail bed.

Grade II: Obliteration of the angle between the nail and nail bed and fluctuation will be present.

Grade III: Parrot beak appearance.

Grade IV: Drumstick appearance and hypertrophic pulmonary periosteoarthropathy (HPPOA) [previously known as hypertrophic pulmonary osteoarthropathy (HPOA)].

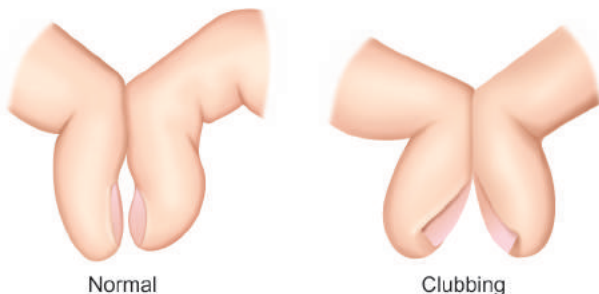
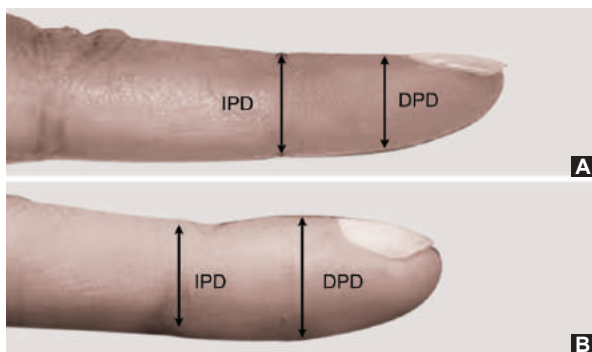


Fig. 3.2: Schamroth's sign

1. Schamroth's sign:

When the dorsal part of the distal phalanges of fingers of both the hands are approximated to each other (Fig. 3.2), in normal subject a diamond-shaped window is formed but in clubbing it will be absent.



Figs 3.3A and B: Interphalangeal depth (IPD) and distal phalangeal depth (DPD):
(A) In normal individual; (B) In case of clubbing

- 2. Interphalangeal depth (IPD) and distal phalangeal depth (DPD):** It is also a useful sign to diagnose clubbing. This can be appreciated by viewing from the side and ask the patient to spread the fingers. In normal individual, the IPD will be more than the DPD (Fig. 3.3A), but in clubbing DPD will be more because of the increased curvature (Fig. 3.3B).
- 3. The profile sign:** Ask the patient to spread the fingers and the examiner has to view from the side (the profile view).

Causes of Clubbing

Respiratory system

- Suppurative lung diseases (lung abscess, empyema, bronchiectasis)
- Diffuse parenchymal lung disease
- Bronchogenic carcinoma
- Metastatic lung cancer
- Mesothelioma
- Sarcoidosis
- Pulmonary AV malformation.

Cardiovascular system

- Congenital cyanotic heart disease
- Atrial myxoma
- Bacterial endocarditis
- Eisenmenger's syndrome.

Gastrointestinal system

- Primary biliary cirrhosis
- Crohn's disease
- Ulcerative colitis
- Gastrointestinal malignancy.

Other causes

- Acromegaly
- Thyrotoxicosis
- Syphilis.

Familial clubbing not common.

Other Less Common Conditions

Unidigital clubbing: Gout, sarcoidosis, local injury.

Unilateral clubbing: Pancoast tumor, brachial AV fistula, aneurysmal dilatation of aorta, subclavian or innominate arteries.

Differential clubbing: Seen with patent ductus arteriosus with reverse shunt—with cyanosis and clubbing of toes and left hand.

Pseudoclubbing: Hyperparathyroidism, occupational (vinyl chloride workers), leprosy and leukemia.

Pedal Edema

Usually, pitting type as seen in congestive cardiac failure, hypoproteinemia and chronic renal failure. Demonstrated by

pressing the finger over the medial malleolus for few seconds and releasing the finger will show pitting. But myxedema and chronic lymphedema due to filariasis are nonpitting.

Jugular Venous Pulse

Engorgement and visible pulsation of neck veins are abnormal above the level of manubrium sterni in sitting up or 45° from horizontal, i.e. semi propped up position. External jugular vein is superficial but internal jugular vein is deep-seated and in direct line with the right atrium, usually the pulsation is seen beneath the sternocleidomastoid muscle.

Patient should be reclined at 45° in good daylight. Patient has to rest his head on a pillow to relax neck muscles and rotate the head slightly away from the side under observation. Look across the neck and identify internal jugular pulsation. Estimate the vertical height in centimeters between the top of jugular pulsation and sternal angle. Mean right atrial pressure is normally less than 7 mm of Hg.

Since the sternal angle is approximately 5 cm above right atrium, the normal JVP should not extend more than 4 cm above the sternal angle.

Hepatojugular reflux—is the distension of the neck veins precipitated by the maneuver of firm pressure over the liver. It is seen in tricuspid regurgitation, heart failure due to other nonvalvular causes, and other conditions including constrictive pericarditis, cardiac tamponade and inferior vena cava obstruction.

Causes of Increased JVP

1. Cor pulmonale.
2. Hyperkinetic circulatory states—fever, pregnancy, hyperthyroidism.

3. Raised intrathoracic or intra-abdominal pressure—obesity, pregnancy, ascites, pleural effusion.
4. Obstructive lesions of vessel—superior vena cava obstruction.

EXAMINATION OF RESPIRATORY SYSTEM

Upper Respiratory Tract

Examination should be done in daylight or with aid of torch. It is preferable to do all components of respiratory examination on a bare chest.

Oral cavity—look for

- Lips—cyanosis, pallor, pursed lip breathing, angioedema, cheilitis, herpes labialis
- Teeth—caries, erosion, anomalies
- Gingivitis—bleeding gums
- Buccal mucosa—leukoplakia, candidiasis, Koplik's spots
- Tongue—pallor, cyanosis, fiery red tongue, stomatitis, leukoplakia, coated tongue, thrush
- Palate—cleft palate, high arched palate, ulcers, tumors
- Tonsils—swelling, abscess
- Posterior pharyngeal wall—congestion
- Uvula—any deviation of uvula.

Nose—saddle nose, use of alae nasi, nasal discharge, epistaxis, deviated nasal septum, polyps, turbinate hypertrophy.

Ears—discharge, discoloration, wax.

Lower Respiratory Tract

Inspection

- **Size, shape and type of chest:**

The normal adult chest is more or less bilaterally symmetrical, ellipsoidal or conical in shape and elliptical

in cross-section. The transverse diameter is greater than anteroposterior diameter (7:5 ratio), the vertical diameter exceeding both anteroposterior and lateral diameters with subcostal angle of 90° (acute in males) (Fig. 3.4).

■ **Chest wall symmetry:**

Symmetrical types of abnormal chest:

- Flat chest—rickets
- Alar chest
- Pectus excavatum—cobble's chest, Marfan's syndrome (Fig. 3.5)
- Pectus carinatum (Pigeon chest)—rickets (Fig. 3.6)
- Barrel chest—emphysema (Fig. 3.7).

Asymmetrical type of abnormal chest:

- Unilateral flattening, retraction, drooping of shoulder (Fig. 3.8), hollowing of supraclavicular and infra clavicular fossae, crowding of ribs or spaces—collapse of lung, fibrosis, wasting of muscles.
- Unilateral bulging—pneumothorax, pleural effusion, any intrathoracic or extrathoracic swelling
- Spinal abnormalities—kyphosis, scoliosis, kyphoscoliosis (Figs 3.9 and 3.10).

All these can later on lead to respiratory or cardiovascular complications.

Look for the presence of:

Any engorged vessels, pulsations; intercostal retractions; movement of subcostal angle; use of accessory muscles and type of breathing.

Inspect the position of:

- Trachea
- Apex beat.

Look for diaphragmatic movement.



Fig. 3.4: Normal



Fig. 3.5: Pectus excavatum



Fig. 3.6: Pectus carinatum



Fig. 3.7: Barrel-shaped



Fig. 3.8: Drooping of shoulder

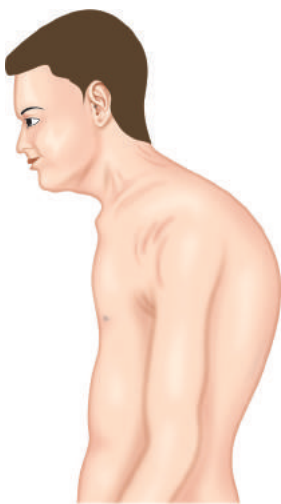


Fig. 3.9: Kyphosis



Fig. 3.10: Kyphoscoliosis

- **Respiratory movements:** Normal respiratory rate 16–20 per minute in adults.

Types of breathing:

- Thoracoabdominal in females
- Abdominothoracic in males.

Abnormalities in respiration:

- Increased rate: Tachypnea in exacerbations of asthma, COPD, tension pneumothorax
- Decreased rate: Narcotic poisoning, diabetic coma, uremia
- Cheyne-stokes breathing: Narcotic poisoning, carbon monoxide poisoning
- Biot's breathing: Meningitis
- Kussmaul's breathing: Diabetic ketoacidosis, metabolic acidosis
- Pursed lip breathing: COPD
- Rapid shallow breathing: Restrictive lung disease, pleuritic chest pain
- Ataxic breathing: Brain stem damage at medullary level
- Apneustic breathing: Brain stem damage at pontine level

- Stridor: Laryngeal spasm, vocal cord paralysis, malignant growth in trachea
- Sleep disordered breathing: Apnea, hypopnea.
- **Thoracic movements**
 - Diminished bilaterally: Emphysema, ankylosing spondylitis, myasthenia gravis
 - Diminished unilaterally: Lung collapse, pleural effusion, pneumothorax, fibrosis
 - Intercostal indrawing: Collapse of lung, fibrosis
 - Hoover's sign: Paradoxical movement of costal margin during inspiration seen in emphysema.
- **Position of apical impulse**

It is half an inch medial to midclavicular line on the left side at the level of 5th intercostal space
- **Sternomastoid sign (Trail's sign)**

Trail's sign (Fig. 3.11): Undue prominence of the clavicular head of sternomastoid on the side to which trachea is

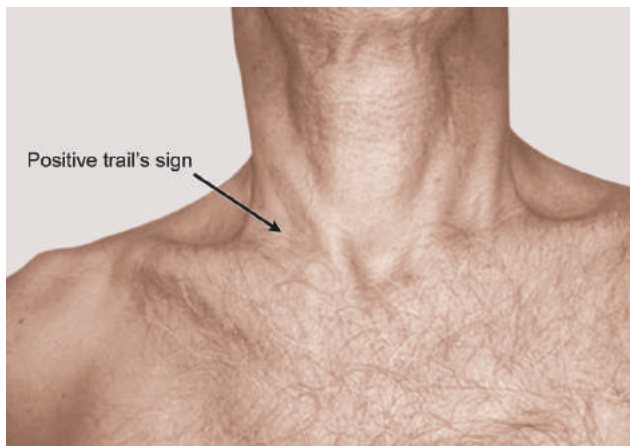


Fig. 3.11: Sternomastoid sign (Trail's sign)

shifted. The pretracheal fascia encloses the clavicular head of sternomastoid on both sides. The clavicular head becomes prominent due to relaxation of pretracheal fascia where the trachea is shifted. Trachea is usually centrally placed but it may deviate to left or right in fibrosis, collapse pneumothorax, pleural effusion fibrothorax and large mass. It indicates the shift of the upper mediastinum

- Distended chest veins, pulsating vessels, sinus and scars.

Palpation

Palpate abnormal pulsation on the chest wall, abnormal swellings—lipoma, sebaceous cyst, tumors involving chest wall.

Confirm inspection findings like:

- **Position of trachea**

If the patient is sitting, stand in front of the patient and fix your ring and index finger on the prominence of the medial end of the clavicle and gently run the middle finger down the trachea. If the trachea is central the finger will come down in a straight line, but any deviation of trachea to left or right, the finger will slide in the direction where trachea is deviated (Fig. 3.12).

Another method is to push gently your finger between the trachea and medial end of sternomastoid muscle and resistance is felt where the trachea is shifted.

- **Apical impulse:**

The impulse is usually felt half an inch (1.25 cm) medial to the midclavicular line in the 5th intercostal space over the left side. Keep the palm of the hand where apical pulsation is seen on inspection with a little tilt of the patient, to usually left side; then locate with the finger for the exact location.

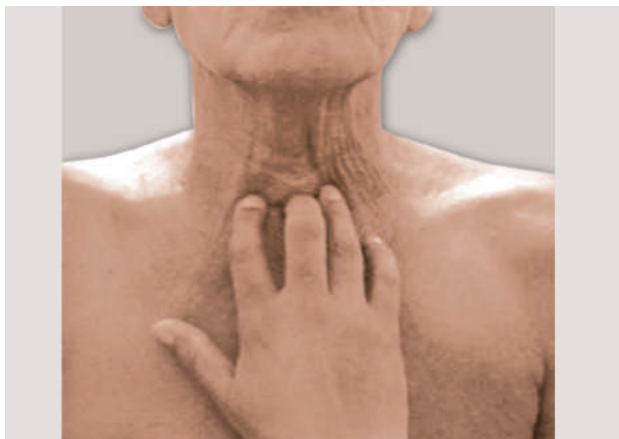


Fig. 3.12: Palpation of trachea

It indicates the shift of the lower mediastinum. Always remember dextrocardia where the apex beat is visualized on the right side.

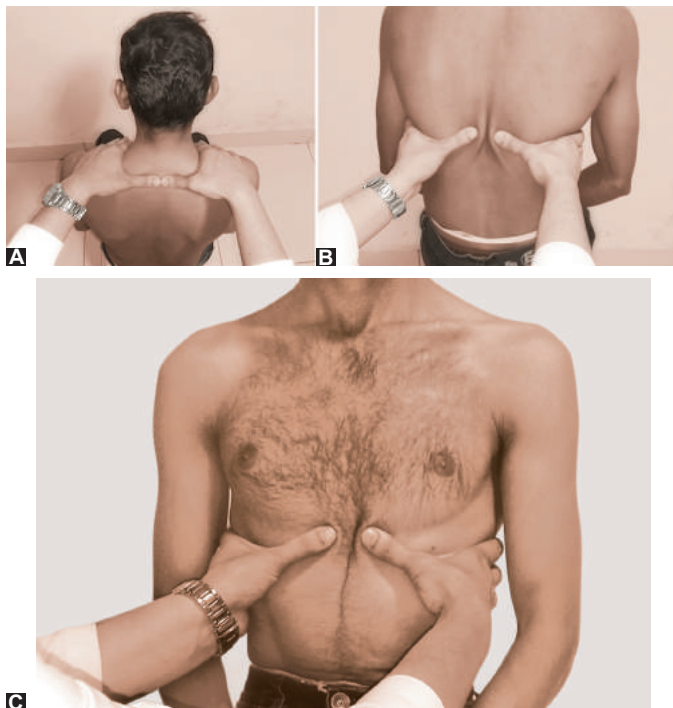
Palpate for widening or narrowing of intercostal space and look for tenderness which may indicate empyema thoracis, subphrenic abscess, fracture, Teitze's syndrome.

■ **Respiratory movements:**

On inspection, respiratory movement is already observed preferably from the foot end and the finding may be subjective if there is not much difference in the movement of left and right hemithorax.

So to confirm, palpation is required and follow these three steps.

For apical region: While the patient is seated with the head bent downwards, the examiner has to stand behind the patient with both the hands placed over the apical region with the thumbs approximated in the midline. The movement (excursion) of the thumbs from the midline



Figs 3.13A toC: Chest wall expansion by palpation:
(A) Apical; (B) Posterior; (C) Anterior

while the patient breathes deep indicates the degree of expansion of the two sides and in case of fibrosis, collapse—the movement may be reduced on the affected side (Fig. 3.13A).

In lying down position, the hands are to be placed over the clavicles and note the movement of the hand while the patient takes deep inspiration. Also the difference in movement can be appreciated by keeping the hands on either side of the neck and assess which hand moves higher.

For the middle and lower regions (interscapular and infra-scapular areas): The patient to be seated with hands crossed over the chest wall. The two hands of the examiner who is standing behind, is placed symmetrically on either side of the patient's chest and this will help to grip the two sides with the palms and thumbs are opposed to meet in the midline. So while the patient is breathing, the thumbs move away from midline normally, but in case of less movement of the thumb on one side, restriction of movement on the affected side is suspected. Patient should be asked to inspire deeply (Fig. 3.13B).

Anterior examination: Keep the palm of both the hands in the 4th or 5th intercostal space, thumbs are opposed to meet in the midline. Ask the patient to take deep inspiration, the thumbs move away from midline normally, but in case of less movement of the thumb on one side restriction of movement on the affected side is suspected (Fig. 3.13C).

In males usually abdominothoracic type of respiratory movement and in females, thoracoabdominal type of respiratory movements are seen. To appreciate this, when the patient is breathing, keep one hand over the chest and other hand over the abdomen. In males, the hand over the abdomen will move first and in females the hand kept over chest will move first.

■ **Measurement of chest expansion:**

The normal circumferences of the chest are taken first with a tape and then the patient is asked to take a deep inspiration and the difference is noted (Fig. 3.14).

Normal expansion is 5–8 cm but in certain conditions it will be less.

Conditions of decreased expansion may be due to pleural effusion, pneumothorax, collapse, fibrosis, etc.



Fig. 3.14: Measurement of chest expansion

Spinoscapular distance is measured from the angle of scapula to the spine and distance is reduced considerably in fibrothorax.

- **Diaphragmatic movement**
- **Tracheal descent with inspiration: (Tracheal tug)**
Cricosternal distance is a good indicator of tracheal descent. The normal cricosternal distance is 4–5 cm. Distance less than 3 fingers during inspiration indicates hyperinflation of lung.
- **Vocal fremitus:**
Usually in a normal subject, low-frequency sound is transmitted to chest wall after it gets filtered by alveoli. In certain diseases it may alter.
When a person produces a sound, there is vibration of chest wall. Larynx vibrates during phonation. The vibrating element is the vocal cords. This vibration is modified by

the mouth, nose, nasal sinus, pharynx and chest cavity (resonators). Thus, sound produced in larynx is transmitted to chest wall as vibration and the examiner can appreciate the vibrations by using the ulnar border of the palm kept on the chest wall to appreciate vocal fremitus.

The patient is asked to say some number in their language (usually one..one..one) which should be distinct and patient is asked to repeat it when the examiner keeps the hand on the chest wall. The spoken words should be clear and with the same intensity.

The words the patient uttered are conducted to the chest wall and the examiner feels the vibration.

Examiner keeps the ulnar border of the hand on the chest wall and tell the patient to say some number and the examiner has to compare symmetrical areas starting from supraclavicular to mammary areas, axillary and infra-axillary areas and suprascapular, interscapular and infrascapular areas (Figs 3.15 and 3.16 for points of reference for percussion).

Vocal fremitus is increased in consolidation, fibrosis and reduced in pleural effusion, pneumothorax, lung collapse.

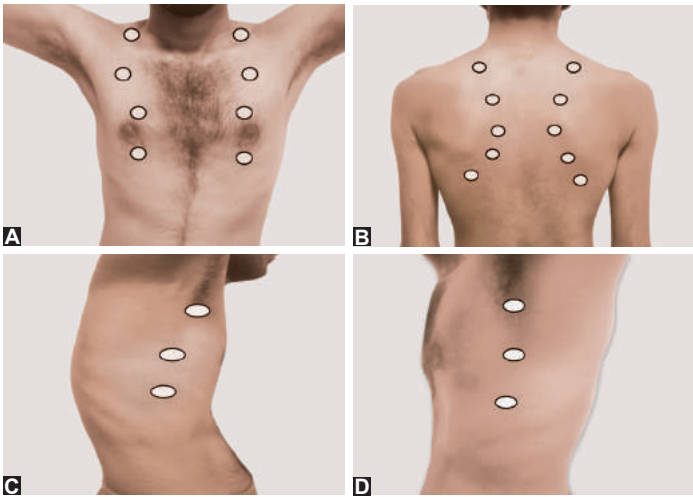
Friction fremitus—palpable rub in pleurisy.

Tactile fremitus —crepitus as in subcutaneous emphysema.

Percussion

By percussing the chest, we will hear the sound produced which is expressed as:

- Normal resonance
- Hyperresonant
- Impaired resonance or dull note
- Stony dull note
- Tympanitic note
- Subtympanitic.



Figs 3.15A to D: Vocal fremitus—topographical areas:
(A) Anterior chest wall; (B) Posterior chest wall; (C) Right lateral; (D) Left lateral



Figs 3.16A to D: Vocal fremitus examination

Percussion is also useful in marking the borders of lung so as to make out whether there is hyperinflation (emphysema) or shrinkage of lung (collapse or fibrosis).

Two points to remember are: Auditory perception of sound elicited and tactile perception of the sense of resistance felt.

Position of patient: Preferably sitting down and not supine because the bed may dampen the sound. But if the patient is in supine position, percussion is carried out in both right and left lateral position.

Start percussion from supraclavicular area in front and from suprascapular area behind.

Anterior percussion:

- Hands on patient's hip (Fig. 3.17).

Posterior percussion:

- For suprascapular percussion, patient's hands are crossed on the thighs and patient is slightly stooping forward but relaxed
- For the interscapular and infrascapular areas, patient is asked to keep the hands crossed on the shoulders (Fig. 3.18).

Lateral percussion:

- Hands over the head.

Cardinal rules of percussion

1. Percussion should be done from resonant to dull areas.
2. The examiner should be close to the patient and carefully listen to percussion note.
3. Plexor finger is the tip of right middle finger which is used to strike the middle phalanx of pleximeter finger kept on the chest wall. Plexor finger acts like a hammer to strike and it should strike the pleximeter finger at right angle (Fig. 3.19).
4. Pleximeter finger—that is the middle finger of the left hand kept firm on the chest wall to avoid air pockets. Only the



Fig. 3.17: Anterior percussion



Fig. 3.18: Posterior percussion



Fig. 3.19: Plexor finger to strike the middle phalanx of pleximeter finger

pleximeter finger should touch the chest wall, not the other fingers.

5. Symmetrical areas should be percussed anteriorly in the left and right interspaces and notes compared. Always keep the pleximeter finger in the interspaces while percussing anteriorly and laterally. On the left side, presence of heart, stomach and spleen and on the right liver may vary the percussion note (which will be explained later).
6. Important to remember is that while percussing only the wrist of the right hand should move and not the elbow.
7. Each stroke must be sudden and withdraw the plexor immediately to avoid dampening of the note.
8. Each stroke should produce a clear audible note and it may be difficult to demonstrate in obese patients.
9. While percussing to demarcate the area of liver or heart, the long axis of the pleximeter finger must be kept parallel to the expected border of the organ.

Direct Percussion

The pleximeter finger is not used but use the plexor to percuss.

Clavicular percussion: May be useful in detecting lesions in the upper lobe. Percussed on the medial third of clavicle. While percussing, the skin over the clavicle should be stretched by the index and middle finger of the other hand.

Sternal percussion: Usually dull note produced by the presence of heart. But in case, the heart is shifted to left or right due to fibrothorax with hyperinflation or collapse, sternal percussion will be resonant.

Areas to be percussed

- Supraclavicular
- Infraclavicular
- Mammary
- Axillary
- Infra-axillary
- Suprascapular
- Interscapular
- Infrascapular.

Kronig's isthmus—is a band area of resonance over each shoulder connecting zones of lung resonance over anterior and posterior aspects of both the sides of the chest apex. It is bounded anteriorly by clavicle, posteriorly by trapezius muscle, laterally by acromion process of scapula and medially by neck structures. It is 5–7 cm in width and is resonant. But in the presence of a tumor or any other abnormality in the apex of upper lobe, it may become dull.

Types of Percussion Note

- Normal resonant note:
 - The normal percussion note in the chest is resonant due to vibration of air in the underlying lung tissue with millions of alveoli, and of low-pitch and clear in character (like tapping an empty metal box)

- Resonant note better heard in the front of chest wall (not in females) than the back. In the axilla, the note may more resonant than other areas. Lesions which are deep-seated (more than 5 cm from the chest wall) and smaller lesion (less than 3 cm in diameter) may not alter percussion note but visible on the X-ray.
- Abnormal:
 - Myotatic irritability (percussion myokymia)—in case of emaciation or wasting of the patient, a percussion stroke over the front of the chest wall produces fibrillatory contraction of the muscle but no note is produced
 - Hyperresonant note—is obtained when the air space in the lung contains more air than normal. Pneumothorax, emphysema (usually bilateral hyperresonance), Compensatory emphysema (unilateral) or a large superficial cavity or emphysematous bulla
 - Tympanitic note—drum-like note normally heard over stomach, intestines. In the chest, this note may be heard in diaphragmatic hernia
 - Subtympanitic note (Skodaic resonance)—hyperresonant note with boxy quality just above the level of pleural effusion. This is produced because of the relaxed lung partially filled with air is pushed upwards and towards the hilum
 - Impaired note—when a part of the lung has no air or less air, it fails to vibrate sufficiently on percussion with slight loss of resonance and called impaired note as happens in consolidation or collapse
 - Dull note—inflammation of lung parenchyma results in collection of fluid, exudates and infiltration of cells in alveoli as in consolidation, which will reduce the resonant note and more muffled than the impaired

note. Also can occur in lung collapse, large tumors and thickened pleura

- Stony dull—the best example is pleural effusion because when examiner percusses over the chest wall practically no sound is heard as if percussing over the thighs or over a stone. Dampening note is due to change in the interphase (the fluid dampens the conduction of vibration to chest wall). But in a pleural effusion above the fluid level, it may be resonant note or subtympaanitic note because of collapsed lung. It can also occur in solid tumor
- Cracked pot resonance—may be demonstrated over a cavity communicating with a bronchus. It occurs when the patient coughs, the air is expelled and examiner may obtain a tympanitic like resonance.

Tidal Percussion

Percussion to find out the lower border of lung resonance on each side on deep inspiration and expiration. This will help in finding out the diaphragmatic movement. This can be done either anteriorly or posteriorly.

‘S’-shaped curve of Ellis

This is useful to find the upper extent of pleural fluid (which is highest in the axilla and lowest in the spine assuming the shape of letter ‘S’). Some authors attribute this to capillary action. In a moderate effusion, lung collapses towards hilum to give space to the fluid to rise in the axilla and this can differentiate hydropneumothorax and encysted effusion where the fluid does not go higher up in the axilla.

Start in front where the stony dullness is present and then go laterally and the examiner has to percuss the extent of dullness in the axilla and then come posteriorly where the dullness is felt. In uncomplicated pleural effusion where the fluid is free,

the stony dull note will be higher up in the axilla indicating the fluid is at a higher level.

Straight line dullness

One of the key points in diagnosing hydropneumothorax and pyopneumothorax. The percussion note does not go up the axilla but the dullness can be elicited as a straight line around the hemithorax and above the dull note there will be hyperresonant note because of air. Other two clinical features for diagnosing hydropneumothorax and pyopneumothorax are shifting dullness and succussion splash (on auscultation)

Shifting dullness

Percussion for shifting dullness: Useful in hydropneumothorax and also to detect small pleural effusion. When there is air and fluid, the fluid shifts to dependent position and the lung and air floats up. The patient is asked to lie down or sit down and start percussing from above down. In the upper part, hyperresonant note may be heard and below where the fluid is present, there is stony dullness. Now turn the patient to lateral decubitus position (if the lesion is on right side ask the patient to lie on left) and wait for sometime before percussion. Now percussion is done, the dull area becomes resonant because the fluid has shifted. Shifting dullness may be absent in pleural effusion. If the examiner suspects pyothorax, the patient has to lie on the lateral side for about 5 minutes or more because the thick pus takes some time to move unlike the pleural fluid.

Cardiac dullness

Along with palpation by percussion cardiac dullness can be elicited.

- Percussion of left heart border:
Ask the patient to lie supine and locate the 5th intercostal space on the left side from the sternal angle. Percuss from midsternal line (usually dull) parallel to the lateral sternal

border and percuss till resonant note heard. This is the left border of heart. If the distance between dull note and resonant note is more than 11 cm it may indicate cardiomegaly

- Percussion of right heart border:

As above patient is to be in supine position. By percussing along the midclavicular line, locate the upper border of liver. Percuss parallel along the right lateral sternal margin and find out the area of dullness. Right heart is enlarged if the dull note is more than 1 cm from the lateral sternal border.

Liver dullness

Percuss anteriorly on the right side from infraclavicular area in the midclavicular line and anterior axillary line and normal liver dullness is heard in the 5th or 6th intercostals space position.

Splenic dullness

Small area of dullness can be demonstrated in the 8th intercostal space in mid-axillary line on the left side due to the presence of spleen.

Grocco's triangle

It is in the form of a triangle bounded medially by midspinal line to the level of 10th thoracic vertebra (lower border of lung resonance), below by horizontal lining extending outwards from 10th thoracic vertebra for about 3–7 cm and laterally by a curved line connecting the above lines. This area may be obliterated in presence of moderate to large pleural effusion over the back of the chest on the contralateral side. This may be due to displacement of posterior mediastinum to the opposite side as a result of fluid collection.

Garland's triangle

Roughly a triangular area where tympanitic note or Skodaic resonance is heard in pleural effusion because of the relaxed lung above.

Traube's space

Semilunar space, bounded by pulmonary resonance, on the right by liver dullness, on the left by splenic dullness and below by the left costal margin. Overlies the stomach contents.

Auscultation

There is variation in airflow in larger airways which is turbulent flow whereas it becomes laminar in smaller airways. The noise of the flow varies from 2000 Hz to 200 Hz and when air reaches the smaller airways and alveoli, filtration takes place and the sound becomes softer (200–400 Hz) and conducted to the chest wall as normal breath sounds previously termed as “vesicular”.

The intensity of breath heard through the chest wall depends on the rate of air flow and the acoustic properties of lung and chest wall. In consolidation, there is no filtration of air by the alveoli and directly conducted to chest wall and the sound heard is similar to that heard over the trachea (tubular breathing), when the sound is reflected at the interface between the lung and any other media like air (pneumothorax) or fluid (pleural effusion).

Auscultation of the lungs includes breath sounds, its character and intensity, vocal resonance and adventitious sounds.

Principles of Auscultation

1. Auscultation should be done in a quiet room, preferably in a sitting position. If the patient cannot assume sitting posture,

roll the patient from one side to the other to examine the back.

2. Ask the patient to take deep breaths through the open mouth.
3. Using the diaphragm of the stethoscope, start auscultation anteriorly at the apices, and move downward till no breath sound is appreciated. Next, listen to the back, starting at the apices and moving downward. At least one complete respiratory cycle should be heard at each site.
4. Always compare symmetrical points on each side.
5. Listen for the quality of the breath sounds, the intensity of the breath sounds and the presence of adventitious sounds.

The examiner has to listen for both inspiratory and expiratory breath sounds in all the topographical areas as described earlier.

Listen carefully for intensity, quality, comparison of inspiratory and expiratory breath sounds and prolongation of breath sounds.

In this book, the authors confine to two types of breath sounds for easy understanding.

1. Normal breath sound (vesicular breath sound).
2. Bronchial breathing of three types:
 - Tubular breathing
 - Cavernous breathing
 - Amphoric breathing.

Vesicular Breath Sound

Vesicular breath sound is a misnomer as vesicles means alveoli and this gives the impression that the breath sound is originating at the alveolar level. However, breath sounds cannot be generated at the alveolar level since airflow is laminar within the alveoli. The expiratory sound is audible only in the early phase. The short expiratory phase is due to the passive nature

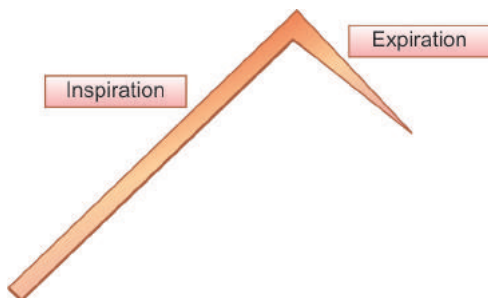


Fig. 3.20: Normal breath sound

of expiration resulting in generation of less turbulent airflow. The origin of both phases of respiration is also in different sites. The inspiratory component originates in the lobar and segmental airways, whereas the expiratory component arises from more central airways.

Characteristics of normal breath sound (Fig. 3.20):

- Rustling of leaves during breeze
- Inspiratory sound louder and greater in intensity than expiratory sound
- Longer duration of inspiratory sound ratio of 3:1
- Absence of pause between inspiration and expiration.

If any change in the normal breath sound is examined, the examiner has to think about an underlying pathology.

Bronchial Breath Sounds

Characteristics of bronchial breath sound (Fig. 3.21) are as follows:

- Expiratory component is louder and longer than inspiratory component
- The ratio between inspiratory and expiratory sound is 2:3
- There is a pause between inspiratory and expiratory sound.

Tubular—normal heard over trachea and this can be kept as reference point to make out whether it is tubular or normal

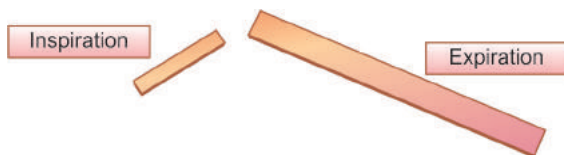


Fig. 3.21: Bronchial breath sounds

breath sound. Tubular breathing is high-pitched in quality heard over consolidation, any mass overlying major airways, above pleural effusion and upper lobe fibrosis.

Cavernous—low-pitched hollow quality usually heard over thick-walled cavity and ‘pulled trachea syndrome’. The cavity should be more than 3 cm in diameter and less than 5 cm from chest wall.

Amphoric—high-pitched bronchial breathing with metallic quality and it resembles blowing through a narrow metal pipe in thin-walled cavity communicating with bronchus, bronchopleural fistula. The presence of amphoric breathing is pathognomonic in diagnosis of bronchopleural fistula.

Adventitious/Added Sounds

Adventitious sounds are additional respiratory sounds superimposed on normal breath sounds. Adventitious lung sounds are classified into two main categories—continuous and interrupted sounds. Interrupted or discontinuous sounds are further classified into fine and coarse crackles and continuous sounds are further classified into wheeze and rhonchi. [International Lung Sound Association]

Wheeze and Rhonchi

Wheezes and rhonchi are continuous musical lung sounds. Continuous adventitious sound lasts more than 250 ms. The American Thoracic Society (ATS) Committee on pulmonary nomenclature defines wheezes as high-pitched continuous

sounds with a dominant frequency of 400 Hz or more, and rhonchi as low-pitched continuous musical sounds with a dominant frequency of about 200 Hz or less.

The mechanism of wheeze:

- Narrowing of airways
- Mucosal edema
- Mucus plugging.

It is also produced by vibration of opposing airways.

Monophonic wheeze—single musical note of constant pitch timing and site during inspiration. The monophonic wheeze may better heard (higher) during expiration than inspiration probably due to tightness of stenosis. Foreign body, malignancies, intramural and extramural compression (e.g. lymph node or mass obstructing the bronchus).

Polyphonic wheeze—it is an expiratory musical sound containing several notes at different pitch throughout expiration as in asthma and chronic bronchitis.

Stridor occurs in both phases of respiration. It is due to narrowing of trachea or larynx.

Crackles (rales, crepitations)

Crackles are discontinuous, explosive and nonmusical adventitious lung sounds normally, heard in inspiration and sometimes during expiration. Crackles are usually classified as fine and coarse crackles based on their duration, loudness, pitch, timing in the respiratory cycle and relationship to coughing and changing body position.

Mechanisms of the production of crackles

Initially, production of crackles was attributed to the passage of air through the accumulated secretions within the large and medium size airways, creating the bubbling sounds.

Forgacs proposed the second theory of crackles. According to Forgacs, small airways that were collapsed during expiration snap open during inspiration as a gradient of gas pressure is developed across the collapsed airways. Sudden explosive opening of the collapsed airways induces a rapid equalization of gas pressures resulting in oscillations of the gas column and development of crackles.

Stress relaxation quadrupole hypothesis

Fredberg and Holford proposed the stress relaxation quadrupole hypothesis of crackles generation. According to this hypothesis, crackles are produced by the vibration in the walls of small airways and not by the air column within airways. Crackles arise due to the sudden opening and closing of airways, resulting in stress waves' propagation in the lung parenchyma.

Liquid bridge hypothesis: Liquid bridges are formed due to abnormal mechanical instabilities in the small airways. Liquid bridge ruptures are responsible for the inspiratory crackle sound and formation of the liquid bridge explains the expiratory crackle.

- Early inspiratory crackles—chronic bronchitis, stage of resolution of pneumonia probably because of reopening of more proximal airways
- Midinspiratory crackles—bronchiectasis, midinspiratory phase is represented from segmental bronchi to terminal bronchioles. They are audible at the mouth and no postural variation and may be altered by coughing. The character may be leathery
- Late inspiratory crackles—interstitial lung diseases, pulmonary edema. Usually heard at the bases. They are produced at small airways. It is due to explosive reopening during the latter part of inspiration of small distal airways

with abnormally opposed walls during the previous expiration. They are not conducted to the mouth and changes with posture. They are called 'Velcro' crackles. Leaning forward will reduce the intensity of the crackles in diffuse parenchymal lung disease, but in pulmonary edema as it takes longer time for the fluid to shift.

Pulmonary squeak

It follows an inspiratory crackle and probably caused by vibration in the wall of a bronchiole as it opens following abnormal closure. May be heard in diffuse parenchymal lung disease and hypersensitivity pneumonitis.

Vocal resonance

The mechanism is same as vocal fremitus.

Usually, low-frequency is transmitted to chest wall after it gets filtered by alveoli in a normal subject. In certain diseases, it may alter.

The sound produced in larynx is transmitted to chest wall as vibration and the patient is asked to say some number in their language (usually one...one...one) which should be distinct and patient is asked to repeat it when the examiner places the diaphragm of the stethoscope on the chest wall. The spoken words should be clear and with the same intensity.

The words the patient uttered are conducted to the chest wall and it will be weak and muffled in normal individuals. The examiner has to compare symmetrical areas starting from supraclavicular to mammary areas, axillary and infra-axillary areas and posteriorly suprascapular, interscapular and infrascapular areas.

Vocal resonance is increased in consolidation, fibrosis and reduced in pleural effusion, pneumothorax, lung collapse.

Pleural rub

Pleural rub is rubbing, grating, leathery sound. It is usually localized and has loud intensity, superficial in character and the sound is intensified when the stethoscope is pressed over the area.

There is a thin layer of lubricating fluid separating parietal pleura from the visceral pleura in a healthy individual. But in inflammation of pleura, the inflamed surfaces rub each other because of the lack of lubricating fluid which produces both pain and friction in a localized area. It is commonly heard in the infra-axillary area but rarely diffuse in nature. Pleural rub is usually heard at the end of inspiration (when the two surfaces rub each other) and at the beginning of expiration when the two layers are in close approximation. Unlike crackles, the rub is not altered with coughing.

It can be differentiated from pericardial rub which is continuous whereas pleural rub is absent when the patient is asked to stop breathing.

Succussion splash

It is a common sign in hydropneumothorax, pyopneumothorax, hemopneumothorax, i.e. when there is air and fluid in the pleural space, succussion splash can be appreciated. Keep the diaphragm of the stethoscope over the fluid-air interphase and shake the patient vigorously. If the sign is positive, a splashing sound is heard. This can be also heard over a large cavity containing fluid and air in the lung and also in herniation of stomach or colon into thoracic cavity through diaphragm. For a clinical diagnosis of hydropneumothorax or pyopneumothorax, the three important signs are: On percussion—shifting dullness, straight line dullness, and on auscultation—succussion splash. The sign can be reproduced by the following method—take a bottle containing water which

is half full and the examiner can appreciate the three signs. Succussion splash can be heard by shaking the bottle.

Hamman sign or mediastinal crunch

It may be heard in case of left-sided pneumothorax. It is a crunching sound heard when there is movement of air during the systolic contraction of the heart. This also can occur in pneumomediastinum, bullous emphysema of the lingular lobe, dilatation of stomach, dilatation of the lower third of esophagus and pneumoperitoneum with the rise in the left diaphragm.

Post-tussive suction

It is heard over a thin wall large cavity in communication with the bronchus. Ask the patient to cough and the air in the cavity will be emptied out, now keep the diaphragm of the stethoscope over the cavity and ask the patient to inspire and the examiner will hear a gushing sound as if air being sucked into the cavity.

Bronchophony

When spoken sounds become more loud or intense and close to the ear while auscultation, it is called Bronchophony. It occurs in consolidation above the level of pleural effusion, lung cavity located superficially. This is due to conduction of the sound directly to the chest wall without filtration taking place in the lung.

Whispering pectoriloquy

To elicit whispering pectoriloquy, the patient is asked to whisper syllables and this may be heard through the stethoscope very clearly as in cavity with communication with the bronchus, consolidation, compressed lung above pleural effusion and obstruction of large bronchus by a tumor proximal to the site of obstruction.

Aegophony/E-A sign

Previously the sounds heard were described as bleating of a goat. The patient is asked to say “Eee” and examiner will hear it as “aa” through the stethoscope. It can be appreciated just above the pleural effusion and consolidation.

Coin test

A coin is placed in the interscapular area and assistant taps the coin with another coin and the examiner auscultates in the anterior chest wall. In case of pneumothorax, a bell-like sound will be heard by the examiner and is called bell tympany.

Scratch sign

The diaphragm of the stethoscope is placed over the sternum. The skin of the patient is scratched at equidistant points from the stethoscope. In pneumothorax, the sound heard will be louder when comparing with the normal side. This is due to high-frequency of sound absorption by the adjacent normal lung.

d’Espine’s sign

Whispered voice heard in the normal individuals over the spines of lower cervical vertebra. But in infancy and childhood, it can be heard below the 7th cervical vertebra and at the level of 3rd thoracic vertebra in adults. When whispered voice is heard below these levels d’Espine’s sign is positive. A positive d’Espine’s sign may be suggestive of enlarged mediastinal or tracheobronchial lymph nodes, tumor in the posterior mediastinum or in central pneumonia.

Common Investigations in Respiratory Evaluation

The main aim of supporting and relevant investigations is to confirm the diagnosis we have already made from detailed history taking and clinical examination.

At present day scenario we have to depend more on these investigations, in case if there is medical litigation to protect ourselves. In a tertiary care center, opinion can be sought from other specialty departments, e.g. cardiothoracic surgery, ENT, etc. Various stains and culture methods are available in microbiology to identify various pathogens. In pathology also various stains are used. The main investigation support is from the radiology department for confirming various diagnoses.

'As medical tests are becoming exorbitantly expensive, basic and cost-effective investigations should be ordered.' In future more supportive tests may be available.

DIAGNOSTIC TESTS

1. **Blood routine**—hemoglobin, total leukocyte count, differential count (especially eosinophils, lymphocytes), erythrocyte sedimentation rate (ESR), tests for HIV (if history of high-risk behavior).

2. **Sputum collection**—by expectoration, laryngeal swabs, gastric aspiration, pharyngeal or tonsillar swabs, tracheo-bronchial aspiration, bronchoalveolar lavage (BAL), induced sputum through inhaled mucolytics (if difficult to expectorate due to thick viscid sputum).

With the sputum, following tests can be performed:

- Acid-fast bacilli staining (AFB)—2 samples; direct smear by ZN stain, LED microscope, NAAT
- AFB culture and sensitivity—if drug resistance suspected along with the other modern techniques (e.g. PCR)
- In bacterial infections—culture and sensitivity
- Fungal hyphae, fungal staining and culture
- Bronchial asthma—eosinophils, plugs, casts, creola bodies, Curschmann's spirals, Charcot Leyden crystals (also in ABPA)
- Asbestos bodies—asbestosis
- Hooklets—hydatid cyst, *Entamoeba histolytica*—hepatopulmonary amoebiasis
- Sputum malignant cytology—carcinoma bronchus.

3. Urine examination (if indicated).
4. X-ray chest (preferably PA view, or AP, lateral, lateral decubitus, Lordotic view, etc. as the condition demands).
5. USG (thorax and abdomen).
6. Fasting and postprandial blood sugar (FBS and PPBS)—preferably for all the patients above the age of 30 years.
7. Paracentesis thoracis in pleural effusion (with all relevant investigations).
8. Pleural biopsy.
9. Lymph node FNAC and biopsy.
10. Liver function testing (LFT—if indicated).
11. Renal function testing (RFT—if indicated).
12. PFT (SPIROMETRY, D_LCO , body plethysmography).

13. Peak flow meter—measure peak expiratory flow rate.
14. ECG and ECHO.
15. Computerized tomography—high resolution (HRCT) or with contrast as the situation demands.
16. ABG.
17. Tuberculin testing—Mantoux test (indicates tuberculous infection and not the disease).
18. Other diagnostic skin testing—(hydatid, sarcoidosis, histoplasmosis, blastomycosis, etc.).
19. Allergy testing—intradermal, sublingual immunotherapy (SLIT).
20. Magnetic resonance imaging (MRI)—if necessary.
21. Barium swallow—in case of dysphagia, but it can be visualized in CT.
22. Bronchoscopy—rigid and fiberoptic bronchoscopy—bronchoalveolar lavage (BAL), biopsy, bronchial brushing, transbronchial biopsy, transbronchoscopic needle aspiration.
23. EBUS.
24. Percutaneous or open lung biopsy.
25. Immunological tests, if indicated—IgG, IgM, IgE.
26. Pleuroscopy.
27. Video-assisted thoracoscopy (VATS).
28. Mediastinoscopy (not popular now).
29. Pulmonary angiography.
30. Ventilation—perfusion scan.
31. PET scan.
32. Polysomnography for sleep-related breathing disorders.
33. Inflammometry: FeNO (bronchial asthma, cystic fibrosis, primary ciliary dyskinesic syndromes), exhaled breath condensate analysis.
34. Transmission electron microscopy (TEM)—primary ciliary dyskinesia.

CERTAIN IMPORTANT TESTS (NOT COMMONLY DONE)

Pulmonary embolism	D-dimer
Inherited emphysema	α 1-antitrypsin
Cystic fibrosis	Specific genetic tests
Lung cancer	Tumor marker (CEA, SCC)
Malignant mesothelioma (mesothelin, fibrillin)	Tumor markers
Pneumonia	Serum procalcitonin
Latent tuberculosis infection	Interferon-gamma release assays (IGRAs)
Sarcoidosis	SACE, serum calcium levels
Hypersensitive pneumonitis	Specific precipitating antibodies
Connective tissue disorders	Rheumatoid factor, ANA, etc.
Unexplained dyspnea (to rule out cardiac cause)	N-Terminal-pro-brain
	Natriuretic peptide
	(NT- proBNP)—increase in heart failure

Now that we have done the physical examination, we will get back to the three sample case studies illustrated in chapter 2 and try to come to a diagnosis.

CASE STUDY I

General Examination

On general examination there was no pallor, icterus, lymphadenopathy and pedal edema. Clubbing grade II present. Body mass index 18.

Upper respiratory tract: Normal.

Lower respiratory tract: Trachea and apex beat appears shifted showing mediastinal shift on inspection.

Palpation: Movements reduced with reduction in chest expansion in the right infraclavicular area. Vocal fremitus increased in the above area.

Percussion: Note impaired.

Auscultation: Bronchial breathing present with coarse crackles with absence of wheeze. Vocal resonance increased.

Provisional diagnosis from history and examination can be—

1. Pulmonary tuberculosis (can be reactivation or fibrosis of right upper lobe).
2. Bronchogenic carcinoma (smoker, scar carcinoma).
3. Bronchiectasis.

To come to a final diagnosis following investigations can be done.

- Blood—hemoglobin, total count differential count, erythrocyte sedimentation rate
- FBS and PPBS (considering the age factor and suspicion of pulmonary tuberculosis)
- Sputum for acid fast bacilli for 2 days
- Sputum culture and sensitivity (especially in bronchiectasis)
- X-ray chest—posterior-anterior view
- High resolution computerized tomography to rule out bronchiectasis and malignancy
- Fiberoptic bronchoscopy in case of malignancy.

If sputum is positive for acid fast bacilli, then invasive or expensive investigations can be avoided.

CASE STUDY II

On general examination: Clubbing was present (grade 3). BMI was 22.

Inspection: Tracheal shift to left and apex beat shifted 2 inches lateral to the midclavicular line towards the axilla.

On palpation: Increased vocal fremitus on the left supraclavicular and infraclavicular areas.

Percussion: Impaired note in the above areas.

Auscultation: Bronchial breathing in the above areas but normal breath sounds in other areas. Crackles heard over the left supraclavicular and infraclavicular areas which are conducted to the mouth.

All the earlier mentioned differential diagnoses (page no. 34) may be applicable in this case. So the battery of laboratory investigations may be extensive.

Investigations:

- Blood—hemoglobin, total count differential count, erythrocyte sedimentation rate
- FBS and PPBS (considering the age factor and suspicion of pulmonary tuberculosis)
- Sputum for acid fast bacilli for 2 days (if sputum is negative for acid fast bacilli) the following tests to be done
- Sputum culture and sensitivity (especially in bronchiectasis)
- X-ray chest—posterior-anterior view
- Liver function test
- High resolution computerized tomography to rule out bronchiectasis and malignancy
- Spirometry
- Fiberoptic bronchoscopy in case of malignancy.

CASE STUDY III

Patient tachypneic with accessory muscles acting but cooperative in giving detailed history. No cyanosis or dehydration. SpO₂ 96%.

Percussion: Bilateral hyperresonant note.

Auscultation: Normal breath sounds with bilateral wheeze.

Probable diagnosis: Acute severe asthma probably exacerbated by secondary infection.

Patient has to be nebulized immediately and IV steroids may be given (detailed treatment of asthma is given later).

When the patient improves, following investigations should be done:

- X-ray chest—posterior anterior view
- Blood—hemoglobin, total count differential count, erythrocyte sedimentation rate
- FBS and PPBS (family history of diabetes mellitus and probably prolonged steroid intake)
- Sputum for acid fast bacilli for 2 days
- Sputum culture and sensitivity
- Once the patient settles spirometry can be done, if sputum for AFB is negative.

Management of Pulmonary Emergencies

ACUTE EXACERBATION OF BRONCHIAL ASTHMA

In a well-controlled asthmatic on medication, a sudden deterioration with increased breathlessness the patient is advised to consult the nearest doctor or hospital. Infections and exposure to allergens are the most important cause of exacerbation. Pneumothorax always should be considered as one of the important factors for increased dyspnea.

Investigations:

- Peak expiratory flow rate (PEFR)
- FEV_1 (if the patient can do it) If the accessory muscles of respiration is in action, it indicates 30% reduction in FEV_1 .
- SpO_2
- PaO_2
- $PaCO_2$
- pH.

Moderate Exacerbation of Asthma

Any one of the criteria:

- Increasing symptoms
- $PEF >50-75\%$ best or predicted or less than 200 L/minute

- Wheezing heard on auscultation
- No features of acute severe asthma.

Acute Severe Asthma

Any one of the criteria:

- PEF 33–50% best or predicted or less than 80 L/minute
- Respiratory rate ≥ 25 /minute
- Heart rate ≥ 110 /minute
- Use of accessory muscles
- Inability to complete sentences in one breath
- Wheezing heard on auscultation.

Life-threatening Asthma

Any one of the criteria:

- PEF $< 33\%$ best or predicted
- $\text{SpO}_2 < 92\%$
- $\text{PaO}_2 < 60$ mm of Hg on oxygen
- $\text{PaCO}_2 > 45$ mm Hg on oxygen
- Silent chest (no wheezes heard because of severe bronchospasm)
- Pulsus paradoxus more than 15 mm of Hg
- Look for pneumothorax
- Cyanosis
- Poor respiratory effort
- Arrhythmia
- Exhaustion, altered conscious level.

Near Fatal Asthma

- Raised PaCO_2 and/or requiring mechanical ventilation with raised inflation pressures.

Criteria for Admission

- Admit patients with any feature of a life-threatening or near fatal attack
- Admit patients with any feature of a severe attack persisting after initial treatment
- Normal or increased PaCO_2 or a decreased pH implies potentially life-threatening situation
- $\text{PaO}_2 < 50$ mm of Hg
- Radiological evidence of pneumothorax, consolidation
- Patients whose peak flow is greater than 75% best or predicted one hour after initial treatment may be discharged from casualty, unless there are other reasons which require admission.

Clinical Features

Severe breathlessness (including too breathless to complete sentences in one breath), tachypnea, tachycardia, wheeze or silent chest (due to acute bronchospasm), cyanosis or collapse.

None of these singly or together is specific and their absence does not exclude a severe attack.

Investigation

PEF or FEV_1

PEF or FEV_1 are useful and valid measures of airway caliber.

Pulse Oximetry

Oxygen saturation (SpO_2) measured by pulse oximetry determines the adequacy of oxygen therapy and the need for arterial blood gas (ABG). The aim of oxygen therapy is to maintain SpO_2 94–98%.

Blood Gases (ABG)

Patients with $\text{SpO}_2 < 92\%$ or other features of life-threatening asthma require ABG measurement.

Chest X-ray

Chest X-ray is recommended:

- Suspected pneumomediastinum or pneumothorax
- Suspected consolidation
- Life-threatening asthma
- Failure to respond to treatment satisfactorily
- Requirement for ventilation.

Treatment

Oxygen

- Give supplementary oxygen to all hypoxemic patients with acute asthma to maintain an SpO_2 level of 94–98%
- Lack of pulse oximetry should not prevent the use of oxygen
- In hospital, ambulance and primary care, nebulized β_2 agonist bronchodilators should be driven by oxygen
- The absence of supplemental oxygen should not prevent nebulized therapy being given if indicated.

β_2 -agonist

Use high dose inhaled β_2 -agonists as first line agents in acute asthma and administer as early as possible. Reserve intravenous β_2 -agonists (more tachycardia) for those patients in whom inhaled therapy cannot be used reliably.

- In patients with severe asthma—that is poorly responsive to an initial bolus dose of β_2 -agonist, consider continuous nebulization with an appropriate nebulizer

- In acute asthma with life-threatening features, the nebulized route (oxygen-driven) is recommended.

Steroid Therapy

- Inhaled steroids
- Intravenous methyl prednisolone 1mg/kg or 40 mg 6th hourly/hydrocortisone hemisuccinate 200 mg stat followed by 100 mg 6th hourly
- Give steroids in adequate doses in all cases of acute asthma. Continue prednisolone 40–50 mg daily for at least 5 days or until recovery.

Ipratropium Bromide

Add nebulized ipratropium bromide (0.5 mg stat and 6th to 8th hourly.) Add to β_2 -agonist treatment for patients with acute severe or life-threatening asthma or those with a poor to initial response to β_2 -agonist therapy (but more useful in COPD).

Other Therapies

- Intravenous methylxanthines—aminophylline (as bolus dosage or drip—blood levels to be kept between 10 and 15 $\mu\text{g/mL}$) or IV deriphyllin
- Consider giving a single dose of IV magnesium sulphate for patients with acute severe asthma who have not had a good initial response to inhaled bronchodilator therapy, life-threatening or near fatal asthma with FEV_1 of 25–30%. IV magnesium sulphate (2 g in 100 mL normal saline IV infusion over 20 minutes) should only be used with caution
- Routine prescription of antibiotics is not indicated for patients with acute asthma
- Heliox
- Intravenous leukotriene antagonists

- Correct dehydration
- No sedation
- Inhaled mucolytics not advised in severe cases as they may worsen cough.

Good Response

1. Sustained response after 60 minutes.
2. Physical examination near normal and patient having no distress.
3. PEF more than 70%.
4. Oxygen saturation more than 95%.

After observing the patient for few hours, he/she can be discharged with the advice to continue with metered dose inhaler containing inhaled corticosteroids and long-acting β_2 -agonist.

Oral steroids to be continued for 7–10 days.

If patient is not responding to the aggressive therapy after 6–12 hours, refer to intensive care.

Refer any patient:

- Requiring ventilatory support
- With acute severe or life-threatening asthma, failing to respond to therapy, evidenced by:
 - Deteriorating PEF (less than 30%)
 - Persisting or worsening hypoxia
 - Hypercapnia
 - ABG analysis showing decreased pH
 - Exhaustion, feeble respiration
 - Drowsiness, confusion, altered conscious state
 - Respiratory arrest.

Intubation

Exhausted patient, with increase in PaCO_2 and developing progressive respiratory acidosis. Initial PaO_2 over 50 mm of Hg

and pH around 7.30 and if the patient is alert and cooperative may not require intubation.

If the patient requires ventilatory support:

- Mode can be either assist control or SIMV
- Tidal volume given should be 5–8 mL/kg
- Initial rate 6–10 breaths/minute
- Peak inspiratory flow rate 70–90 liters/minute
- End inspiratory plateau pressure to be kept below 35 cm of H₂O
- PEEP adjusted to 75–80% of measured auto PEEP level
- FIO₂ to be kept to maintain PaCO₂
- Sedation to prevent tachypnea and allow the patient to rest during the first 24 hours.

Permissive hypercapnia protects the lung from ventilator-induced lung injury by using smaller than usual tidal volumes and limiting alveolar distending pressure.

β_2 -agonist therapy to be continued during mechanical ventilation with higher doses because of the deposition of drug on the walls of endotracheal tube.

Complications expected are—barotrauma, prolonged muscle weakness due to high doses of steroids and muscle relaxants.

The patient is discharged with the advice to continue with metered dose inhaler containing inhaled corticosteroids and long-acting β_2 -agonist.

Oral steroids to be continued for 7–10 days.

Patient should be advised to use peak flow meter in the morning and evening and if there is a fall advised to approach the treating physician.

Nebulizer Solutions Available

- Salbutamol: 1 mL of salbutamol solution contains 5 mg of salbutamol

- Budesonide: 2 mL solution contain 0.5 mg
- Ipratropium: 250 mcg/mL
- Fluticasone: 0.5 mg in 2 mL
- Combination of Formoterol 0.5 mg and Budesonide 1 mg in 2 mL
- Combination of Ipratropium (500 mcg) and Levosalbutamol (1.25 mg) in 2.5 mL.

In ultrasonic nebulizers, only solution can be delivered and not suspension (Budesonide is available in suspension form).

ACUTE EXACERBATION OF COPD

In a COPD patient, the precipitating factors can be infections, weather changes, nonadherence to medications, inhalation of tobacco smoke, air pollution, etc.

Sudden onset of symptoms like:

- Increase in cough
- Increase in breathlessness
- Increase in sputum volume and change in its color (white to green, yellow or blood-streaked)
- Fever
- Chest pain
- Increased tiredness
- Increase in oxygen requirement (for those on long-term oxygen therapy)
- Altered sensorium.

Usually, patients present with varying combination of these symptoms. During clinical assessment, objective evaluation is necessary to assess severity of underlying COPD, the presence of comorbidities and history of previous exacerbation. The physical examination should evaluate the effect of the episode on hemodynamic and respiratory systems.

Investigation

Look for the following:

- Use of accessory muscles
- Paradoxical chest movements
- Central cyanosis
- Systolic pressure less than 90 mm of Hg
- Respiratory rate 30/minute or more
- Heart rate >110/minute
- Altered mental status
- Asterixis (flapping tremor)
- Severe comorbid conditions
- SpO₂ less than 90%.

Exclude:

- Pneumonia
- Pneumothorax
- Pulmonary embolism
- Paroxysmal atrial tachycardia (arrhythmias)
- Pulmonary edema
- Pleural effusion
- Associated cor pulmonale.

Presence of any one of the above mentioned signs is an indicator for hospital admission.

The diagnostic procedures to perform, such as chest radiology, sputum and blood examination, ABG and serum electrolytes. Spirometry may not be possible.

Treatment to Follow

- Bronchodilators
- Steroids
- Antibiotics
- Oxygen therapy
- Mechanical ventilation—noninvasive or invasive.

TENSION PNEUMOTHORAX

A tension pneumothorax is a complete collapse of the lung. It occurs when air enters, but does not leave the pleural space.

Causes

Any condition that leads to pneumothorax can cause a tension pneumothorax. In uncomplicated pneumothorax, air can enter and leave the pleural space easily. In tension pneumothorax, however, air enters the pleural space with each breath and gets trapped there.

As the amount of trapped air increases, pressure builds up in the chest. The lung collapses on that side and can push the important structures in the center of the chest (such as the heart, major blood vessels and airways) towards the other side of the chest. The shift can cause the other lung to become compressed, and can affect the flow of blood returning to the heart and the lung excursion of the opposite side also is compromised.

This situation can lead to low blood pressure, shock and death.

Symptoms

- Sudden chest pain
- Dyspnea
- Chest tightness
- Cyanosis
- Tachycardia
- Hypotension
- Decreased mental alertness
- Tachypnea
- Bulging (distended) veins in the neck
- Profuse sweating.

Examination

Patient in distress with the above symptoms as it is a medical emergency. If suspected, percussion (hyperresonant note) and on auscultation the breath sounds are decreased. Mediastinal shift may be noted. There may be subcutaneous emphysema.

Tests used to diagnose tension pneumothorax include:

- A chest X-ray
- Arterial blood gases
- Electrocardiogram.

Treatment

Treatment is aimed to remove the air from the pleural space, allowing the lung to re-expand. In an emergency, a small needle (such as a standard intravenous needle) may be placed into the chest cavity through the ribs to relieve pressure.

The standard treatment is an intercostal tube with water seal. The chest tube is attached to a vacuum bottle that slowly removes air from the chest cavity. This allows the lung to re-expand. As the lung heals and stops leaking air, the vacuum is turned down and then the chest tube is removed. Sometimes, it can take several days for the affected lung to fully re-expand.

Pleurodesis—if the problem happens again, or if the lung does not re-expand after 5 days with a chest tube in place.

Prognosis

Up to 50% of patients who have a pneumothorax will have another. There are no long-term complications after a successful treatment.

Possible Complications

- Acute respiratory failure
- Air in the mediastinal space, which can interfere with heart and lung function (pneumomediastinum)
- Very low blood pressure (shock)
- Death.

HEMOPTYSIS

Hemoptysis is coughing of blood.

Identify which Side is Bleeding

By past history of respiratory illness and treatment usually volunteered by the patient and by auscultation (e.g. post-tubercular right upper lobe bronchiectasis)—presence of crackles, old X-rays.

Quantification is given in the earlier chapter.

Position

Patient whose right lung is bleeding should be placed in the right side down decubitus position (with head end low), whereas a patient whose left lung is bleeding should be placed in the left side down decubitus position (with head end low). The purpose of these positions is to protect the nonbleeding lung, since spillage of blood into the nonbleeding lung may prevent gas exchange by blocking the airway with clot or filling the alveoli with blood.

Guidelines for the Management of Hemoptysis

Initial Management

Airway

Establishing patent airway by intubation

- Breathing

- Circulation
If the patient cannot maintain his/her airway—intubation
- Make the patient lie on the same side as lesion/abnormality on CXR
- IV access and volume replacement
- Hemostatic (role controversial)
- Blood for cross-match, complete blood count and clotting time
- Hemoptysis: >150 mL/airway compromise.

Further Assessment

- Bronchoscopy
- CT thorax for anatomical localization.

Management

- Bronchial arterial embolization/surgery/bronchoscopy (rigid or flexible) to identify source of bleeding
- Assume infection in patients with CF/bronchiectasis and treat aggressively with antibiotics.

RESPIRATORY FAILURE

'All patients require ABG, ECG and X-ray chest.'

Respiratory failure occurs when pulmonary gas exchange is impaired resulting in hypoxemia ($\text{PaO}_2 < 60 \text{ mm Hg}$) with or without hypercarbia ($\text{PaCO}_2 > 55 \text{ mm Hg}$).

Classification

- Type I or Hypoxemic respiratory failure
- Type II or Hypercapnic respiratory failure
- Type III or Perioperative respiratory failure
- Type IV or Respiratory failure associated with shock.

Type I Respiratory Failure

- Characterized by an arterial oxygen tension (PaO_2) lower than 60 mm Hg with a normal or low arterial carbon dioxide tension (PaCO_2)
 - This is the most common form of respiratory failure
 - There are two mechanisms described in the pathophysiology:
 - Ventilation—perfusion mismatch
 - Right to left shunt
 - The common clinical features include sweating, restlessness, anxiety, tachycardia and tachypnea
 - This is seen in various conditions like pneumonia, pulmonary edema, pneumothorax and pulmonary emboli
 - Management:
 - Maintain a patent airway
 - Supplementary oxygenation
 - Principles of ventilatory support—administration of PEEP
 - Identification and treatment of the primary condition.
- (For further details refer prescribed textbooks).

Type II Respiratory Failure

- Also called ventilatory respiratory failure or pump failure
- It is characterized by a PaCO_2 higher than 55 mm Hg
- Hypoxemia is also common in patients with hypercapnic respiratory failure
- The mechanism of pathophysiology is alveolar hypoventilation.

This may be due to:

- Reduced ventilatory effort
- Inability to overcome an increased resistance to ventilation

- Failure to compensate for increase in dead space or increased carbon dioxide production.

The common clinical features include sweating, bounding pulse, tachycardia, headache, flapping tremors and drowsiness.

It can be further classified into:

- Acute
- Acute on chronic
- Chronic.

Acute:

It refers to an acute rise in PaCO_2 level, a rise in H^+ level and a fall in pH level.

Acute on chronic:

There is a fall in pH level due to acutely increased PaCO_2 level associated with chronic elevated HCO_3^- levels.

Chronic:

Normal pH levels associated with chronically elevated PaCO_2 and HCO_3^- levels.

- Acute and acute on chronic types are seen in acute exacerbations of COPD
- Chronic type is seen in conditions like COPD, obesity hypoventilation syndrome and motor neuron disease.

Management

- Maintain a patent airway
 - Principles of ventilatory support—administration of pressure support
 - Identification and treatment of the primary condition.
- (For further details refer prescribed textbooks).

Type III Respiratory Failure

- Occurs as a result of lung atelectasis and is seen commonly in the perioperative period

- After general anesthesia, decrease in functional residual capacity leads to collapse of dependent lung units and subsequent hypoventilation.

Management

- Frequent change in position
- Chest physiotherapy
- Adequate analgesia
- Principles of ventilatory support—administration of PEEP to reverse regional atelectasis.

Type IV Respiratory Failure

- Occurs due to hypoperfusion of respiratory muscles in patients with shock
- Patients in shock suffer respiratory distress because of hypoperfusion resulting in lactic acidosis.

Management

- Correction of shock
- Invasive mechanical ventilation (noninvasive ventilation is contraindicated as PEEP may worsen the hypotension).

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